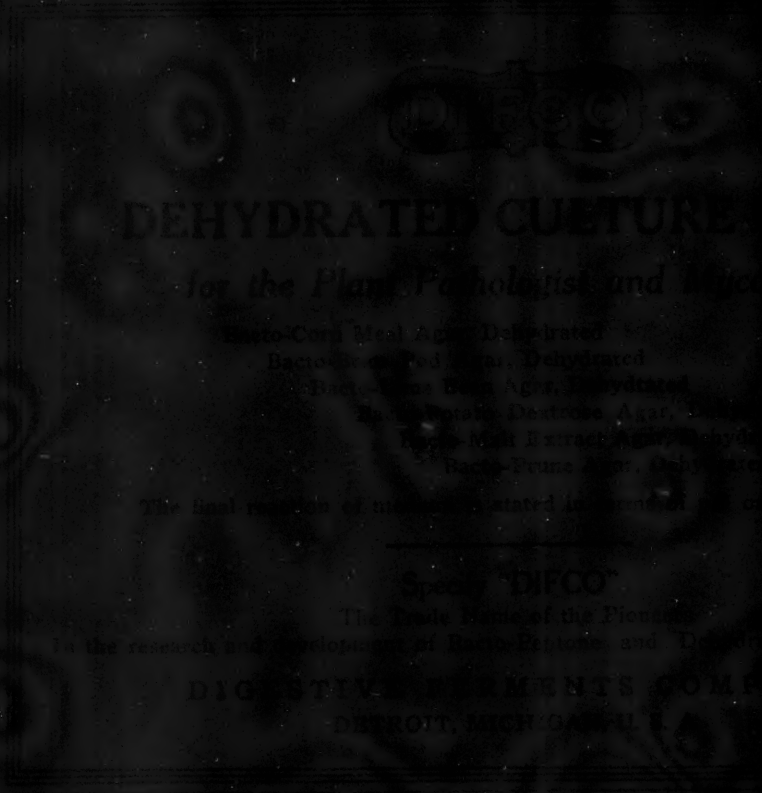

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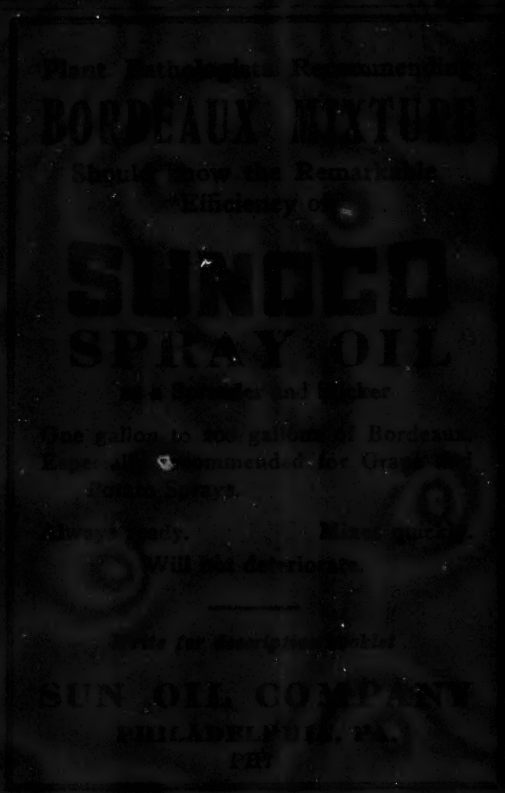

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PHYTOPATHOLOGY

VOLUME XVII NUMBER 8

AUGUST, 1927

COMMERCIAL TOBACCOS AND CURED LEAF AS A SOURCE OF MOSAIC DISEASE IN TOBACCO [1]

W. D. VALLEAU AND E. M. JOHNSON

The question of the source of the initial mosaic infections of tobacco in the plant bed and the field has not been satisfactorily answered, nor has the source been ascertained of sporadic cases of the disease in experimental and commercial greenhouses, where tomatoes and other susceptible plants are being grown. Weed hosts are usually quite prevalent in tobacco sections of Kentucky and may be considered an important source of infection. However, when they are completely removed from a plant bed and plants from the bed are set in the usual manner, extensive infection may be evident soon after growth commences. Because tobacco plants are handled so much in the operations of weeding, transplanting, worming, suckering and topping, the personal habits of the workers, with respect to the use of tobacco, should be given careful consideration in their relation to the introduction and spread of the disease.

The following studies were made with the object of determining to what extent the use of tobacco by workmen might be responsible for at least a part of the mosaic infection in the plant bed and the initial infection in the field, and they may serve to explain many cases of mosaic infection in greenhouses where an obvious source of infection did not appear to be present.

After several seasons of observation of plant beds on the Experiment Station farm, we are of the opinion that mosaic rarely if ever develops in plant beds which have been sowed and then not touched by man; whereas in beds which have been weeded by the average farm hand the disease is likely to be found on an occasional plant and following the first pulling may be widely distributed in the bed. This might be explained on the basis of the presence of occasional weed carriers, but from the results obtained where care has been used not to introduce the disease on the hands of the workers, it would seem that man may be as important a source of infection

[1] Published with the approval of the Director of the Kentucky Agricultural Experiment Station.

as the weed hosts. For example: in a rotation, one field of which is set to about 13,000 tobacco plants each year, records have been kept of the initial mosaic infection since 1922. In 1922, 1923, and 1924, with no precautions other than pulling suspicious weeds from the plant bed some time before pulling plants, the mosaic was 9 per cent, 6 per cent, and 8.7 per cent, respectively, after growth commenced following transplanting. In each of these years men pulling plants used "natural leaf" chewing tobacco. In 1925 the mosaic infection was only 2.1 per cent; two of the men who pulled plants used no tobacco, while one chewed plug tobacco. This precaution was taken to prevent possible angular leaf-spot infection from entering the bed from "natural leaf" chewing tobacco. Toward the end of the planting one of the men who had not chewed was given permission to chew five-year-old "natural leaf" tobacco, and following this the initial mosaic infection in the field rose to 16 per cent. Subsequent tests of this old tobacco indicated that it carried mosaic, as 100 per cent of the plants inoculated with a decoction of it developed mosaic. In 1926 the men who pulled and set were furnished with "natural leaf" and plug tobacco sterilized in the autoclave with the object of destroying both leaf-spot organisms and mosaic. The initial mosaic infection in the field was 0.44 per cent.[2] Three weeks later no mosaic was found in the bed from which these plants were pulled. One end of this bed was located within a few feet of the tobacco field where tobacco had been growing in rotation for several years, thus offering a good opportunity for the weed hosts in the neighborhood to become infected. Bull nettles and ground cherries are very prevalent all about this field. The results suggest that the weed hosts have not been an important factor in initial infection in the years when infection was high.

Another bed on the Experiment Station farm, in which 166 different strains of tobacco were grown and set in the field, was found to be free from mosaic at setting time and three weeks later. Particular care was taken to prevent the introduction of any mosaic on the hands: the plants were pulled by the writers, who washed their hands before each lot of plants was pulled. In spite of these precautions, 0.44 per cent infection resulted in one field of 3,000 plants, and 0.016 in another of 5,500 plants. There was no infection in a third field of about 2,000 plants. Each field had been used for tobacco continuously. From the same bed a block of 20,800 plants from 53 separate pullings (variety test) had developed 0.13 per cent mosaic 25 days after setting; a block of 2,000, 1.05 per cent, and another block of 3,700 plants was entirely free from mosaic. In all cases except the one in which 1.05 per cent mosaic developed, the infected plants were scattered

[2] In 1927, under the same conditions, the initial infection in 13,287 plants was .06 per cent.

and infection appeared not to be associated with pulling. In the one case infection was largely limited to two varieties and might have been associated with pulling. The source of these infections is not known. Six isolated beds of plants in a wood were still free from mosaic in the fall, and ten isolated plots of tobacco set from them by the writers were free from infection with the exception of one plant which grew within a few inches of a diseased ground cherry. These plants were set in a field which had not grown tobacco for many years.

At the western Kentucky substation, where the men were not allowed to use any tobacco while setting and washed their hands before handling plants, no mosaic had developed in extensive plantings several weeks after setting. In experimental plots where plants were pulled and set by men using "natural leaf" chewing tobacco, the mosaic infection was 33.8 per cent; in alternating check plots pulled and set by men with clean hands and not using tobacco, the infection was 9 per cent. The counts were not made until July 19, when the plants were more than knee high. Cultivation had been across the plots, giving an opportunity for spread from row to row. These results, although by no means conclusive, suggest strongly that the human factor is an important one in initial mosaic infection, and suggest that, by eliminating as nearly as possible the virus from the hands of workers, initial mosaic infection may be greatly reduced.

Clinton[3] showed several years ago that if tobacco trash were used as a plant bed fertilizer, mosaic might be expected to develop. The idea, therefore, of cured tobacco acting as a source of infection is not new. Tests of random samples of cured Burley tobacco show that they are usually capable of causing infection in healthy plants.

EFFECT OF AGE ON TOBACCO MOSAIC VIRUS

Age seems to have but little effect on the virus in cured tobacco as indicated by the five-year-old samples above mentioned. Further tests on the effect of age on the virus have been made with samples collected by the Chemistry Department of the Kentucky Experiment Station and carefully preserved in covered glass jars properly labeled. These were kindly furnished by Dr. A. M. Peter. These samples ranged in age from 15 to 30 years when first obtained by us. The early inoculations were made by wetting the ground tobacco with water and rubbing it on the leaves with the fingers. The hands were washed between each inoculation with soap and running water and nothing but the plant was subsequently touched, the decoction being poured on the hands of the operator by another person. In these tests of old material, positive results were obtained from material 15 to 16 years old, 17 to 18, 23, 26 to 27, and 29 to 30 years old.

[3] Clinton, G. P. Report of the station botanist. Conn. Agr. Exp. Sta. 1914.

In a later test the plants were not touched at all with the hands, but each individual plant was inoculated with a separate decoction applied with individual swabs of cheesecloth on sticks, the leaf being held with a piece of waxed paper, changed for each plant. All implements including glassware and swabs were sterilized under high steam pressure before using or by boiling 15 minutes in water, leaving no apparent source of accidental infection. Several leaves on each plant were rubbed to increase the chance of infection. The sixth and seventh plants throughout the series were inoculated with the 31-year-old sample, steam sterilized 15 minutes at 10 pounds pressure, making 18 plants in all which were held as checks. These were placed at intervals between the other plants in the bench. Insects were kept in control by cyanide fumigation. The results of this test, in which there appeared to be no source of accidental infection, are given in table 1.

TABLE 1.—*The results of inoculating tobacco plants with decoctions from ground tobacco of different ages*

No. plants inoculated	Age of inoculum in years	No. plants infected	Incubation periods in days	Type of mosaic
6	16	1	13	Severe
6	17	2	13, 13	Not recorded
6	18	5	6, 8, 9, 9, 9	1 severe, 4 mild
6	20	6	8, 8, 9, 9, 9, 10	Mild
6	24	0	.....	.....
6	28	0	.....	.....
6	30	1	8	Mild
6	31	3	8, 13, 16	Mild
18	Mosaic tobacco sterilized	0	.....	.....

These results seem to show conclusively that tobacco samples kept in closed jars in a relatively dark room for periods of 16 to 31 years may still be viruliferous.

Two types of mosaic resulted from these inoculations. The majority of infections were of the mild type in which a mosaic pattern is clearly evident although the contrast in color is not great and the leaves are not distorted. There was also a more severe type in which the pattern is striking and the leaves usually distorted. These two types are commonly present in Kentucky tobacco fields. In one case (18-year-old inoculum) both types resulted. In all cases the inoculum used consisted of a composite of samples collected the same year, so the appearance of both types of mosaic is not surprising. The fact that the mild type predominated does not necessarily

mean that it is longer lived than the severe type but suggests that in selecting samples for analyses only those were taken which appeared healthy. As the more severe mosaic causes much more prominent symptoms than the mild, the chances of plants being selected which were infected with severe mosaic were comparatively small.

MOSAIC IN "NATURAL LEAF" CHEWING TOBACCO

Although it is by no means certain that all tobacco is infected with mosaic at cutting time, yet it may be assumed that following the operations of worming, suckering, and topping, comparatively few plants remain free from the disease. This assumption is borne out by observations of the disease in the second crop of suckers.

A large majority of Kentucky tobacco growers chew tobacco taken at random from the cured leaf and made into twists. As this tobacco receives no further treatment, it is almost certain that any of it may contain the mosaic virus. As a result of handling the tobacco in a dry state and of wiping the mouth while chewing, the hands are almost certain to become contaminated with the virus. Weeding and pulling plants for setting is usually done while the plants are wet, thus affording ideal conditions for inoculation of an occasional plant during these operations if the hands have become contaminated. Although the facts cited earlier in this paper indicate that this source of mosaic infection may be an important one, another example is cited based on an experiment with tomatoes conducted by the writers. A small portion of a steamed tobacco bed was sowed to tomatoes. Solanaceous weeds had been dug from the bed in previous years. On June 9, 1926, 28 tomato plants were pulled while tobacco was being chewed which was known to be viruliferous. The mouth was wiped from time to time during pulling so that the fingers became contaminated with saliva. These plants were set, the hands washed in soap and water, and 28 more plants pulled from the same bed and set in the next row. In 16 days, 21, or 75 per cent, of the plants pulled with contaminated hands were showing positive signs of mosaic, while the check plants were all healthy 36 days after setting.

MOSAIC IN COMMERCIAL TOBACCO

Considering the almost constant presence of mosaic in tobacco fields, and in view of the longevity of the virus in dry tobacco, commercial tobaccos must all be looked on with suspicion as possible carriers of mosaic. It is probable that a more extensive knowledge of the manufacturing processes used in the preparation of certain brands of tobacco will automatically eliminate them as possible carriers of the disease. For example, we have

TABLE 2.—*The effect of inoculating Turkish tobacco plants and tomatoes with decoctions of commercial brands of tobacco. The inoculations were made in the order given*

Brand[a]	No. plants inoculated	No. plants mosaic	Incubation periods in days
Experiment I			
Reynolds' Natural Leaf (1), plug	5	3	15, 17, 19
Yellow Tag, do	3	0	.....
Apple Suncured (1), do	3	1	12
Old Kentucky, do	3	0	.....
Penn's Red J., do	3	0	.....
Star, do	3	0	.....
King Pin, do	3	0	.....
Futurity, twist	3	0	.....
Brown's Mule (1), plug	3	1	17
Climax (1), do	3	3	13, 17, 23
Richland, twist	3	0	.....
Strater's Natural Leaf, do	3	0	.....
Drummond (1), plug	3	0	.....
Hancock's Fig, do	3	0	.....
Checks (Clean hands and water)	2	0	.....
Experiment II[b]			
Apple Suncured (1), plug	1	0	.....
Old Kentucky, do	2	0	.....
Penn's Red J., do	3	0	.....
Star, do	3	0	.....
Brown's Mule (1), do	2	0	.....
Strater's Natural Leaf, do	3	0	.....
Drummond (1), do	3	0	.....
Sterile tobacco decoction (boiled)	1	0	.....
Experiment III			
Reynolds' Natural Leaf (1), chewed, hands wet with saliva and this rubbed on plants when set	3	0	.....
Experiment IV			
Kate Gravely, plug	3	0	.....
Spark Plug, do	3	0	.....
Masterpiece, do	3	0	.....
Buster, do	3	0	.....
Penn's Pen., do	3	0	.....
Maritiana, do	3	0	.....
Tinsley's Natural Leaf, do	3	0	.....
Boot Jack, do	3	0	.....
Check	3	0	.....

TABLE 2 (*Continued*)

Brand[a]	No. plants inoculated	No. plants mosaic	Incubation periods in days
Experiment V			
Days Work, plug	5	0	.....
Schnapp's, do	5	0	.....
Fish-hook (1), do	5	0	.....
Climax (2), do	5	0	.....
Brown's Mule (2), do	5	0	.....
Star, do	5	0	.....
Apple Suncured (2), do	5	0	.....
Composite of Old Loyalty, Five Bros., Bull Durham, Stud, Duke's Mixture and Granger (rough cut), Granulated Smoking	5	2	8
Experiment VI			
Composite of Tiger, 8-hour Union, Pay Car, Beechnut and Mail Pouch	5	0	.....
Bruton's Scotch Snuff	5	0	.....
Dark tobacco 5-years old, not commercial	5	5	7, 7, 7, 9, 11
Chesterfield cigarettes (1)	5	1	11
Fresh mosaic material	3	3	5
Experiment VII			
Climax (1), chewed and saliva rubbed on hand and plants pulled and set	3	3	21, 25, 25
Checks, (clean hands)	3	0	.....
Experiment VIII[c]			
Lucky Strike (1), cigarette	3	2	11, 15
Camel (1), do	3	1	11
Chesterfield (2), do	3	3	8, 10, 10
Reynolds' Nat. Leaf (2), plug	3	1	14
Apple Suncured (3), do	3	2	10, 14
Brown's Mule (3), do	3	0	.....
Drummond (2), do	3	0	.....
Reynolds' Nat. Leaf (3), do	3	0	.....
Brown's Mule (4), do	3	0	.....
Drummond (3), do	3	0	.....
Apple Suncured (4), do	3	2	9, 16
Reynolds' Nat. Leaf (4), do	3	2	16, 35
Drummond (4), do	3	0	.....
Brown's Mule (5), do	3	1	19
Apple Suncured (5), do	3	1	9
Climax (3), do	3	0	.....
Checks (rubbed with juice from healthy plants)	3	0	.....

TABLE 2 (*Continued*)

Brand[a]	No. plants inoculated	No. plants mosaic	Incubation periods in days
Experiment IX			
Old Loyalty, gran. smoking	3	3	9, 13, 17
Five Bros., do	3	3	13, 14, 15
Stud, do	3	3	13, 14, 15
Bull Durham, do	3	3	9, 13, 14
Duke's Mixture, do	3	3	12, 14, 17
Granger Rough Cut, plug cut	3	0	.....
Check (rubbed with one of own leaves)	3	0	.....
Experiment X			
Fish-hook (1), plug	3	0	.....
Mosaic tobacco steamed 105° C. 10 min.	3	0	.....
Tomato Plants			
Chesterfield (2), cigarette	3	2	25, 25 (suspicious in 9 days)
Reynolds' Natural Leaf (1), plug	3	?	Appeared very mild in 3 plants
Climax (1), do	3	?	Appeared very mild in 3 plants
Camel cigarettes (1)	3	1	25 (suspicious in 9 days)
Experiment XI			
Chesterfield cigarettes (3)	3	3	.....
Check	1	0	.....
Experiment XII[d]			
Chesterfield (4), cigarette	5	2	16, 17
Camels (2), do	5	0	.....
Lucky Strike (2), do	5	3	8, 9, 9
Herbert Tareyton, do	5	2	9, 9
Piedmont, do	5	3	12, 14, 18
Fatima, do	5	5	5, 9, 13, 14, 14
Melachrino, do	5	5	9, 9, 9, 13, 14
Checks	14	0	.....

[a] The number following the brand indicates the particular sample of this brand used.

[b] Plants inoculated with a concentrated decoction, the decoction being injected into several parts of the plants with a hypodermic needle. Plants which remained healthy in the previous test used again with the same brand of tobacco.

[c] The plants of the above series which remained healthy were again inoculated with a second decoction of their respective plugs and again remained healthy.

[d] A separate cigarette, swab, etc., was used for each of the above plants. Two check plants were used for each brand of cigarettes, one being inoculated with leaves from two and the other with leaves from three of the five plants to be inoculated. This is a check on the method of inoculation and also demonstrates that the plants used were free from mosaic when the experiment was begun.

failed to obtain infection, except in one plant, when manufactured twists have been used as the source of inoculum. One manufacturer informed us that it had been found necessary to steam the twists before packing for shipment to prevent spoiling.

Before storing in hogsheads, Burley tobacco is run through a redrier at temperatures ranging from 140° F. to 170° F. for 40 minutes. This has no apparent effect on the virus, as 18 plants inoculated with tobacco redried at 150° F., 18 with tobacco redried at 165° F., and 18 with tobacco not redried, all gave 100 per cent infection, while the checks remained healthy. The samples used were selected at random at the redrying plant and represent the results which may be expected from cured Burley leaf in general. Burley tobacco, therefore, when purchased by the manufacturer, is viruliferous and, if it goes through no processes which destroy the virus during manufacture, should be so in the final product. The same is probably true of other types of tobacco.

A number of brands of commercial tobacco have been tested in the course of these studies to see to what extent they may carry the virus. Preliminary inoculations were made with plug tobaccos which gave positive results in some cases, but the results are open to question as the plugs were not flamed and may therefore have carried small particles of infected tobacco, as dust, into the decoction. Later inoculations, recorded in table 2, were made with greater care; tools were sterile, and the plugs of tobacco were always carefully flamed. A portion of the plug was then shaved off onto a clean piece of letter paper and poured from this into an Erlenmeyer flask containing a small amount of sterile water. The inoculations were made by the finger-rubbing method, the hands being washed with soap and running water between inoculations. The person making the inoculations touched nothing but the soap, water, plants, and decoction during any set of inoculations, another person shifting the pots, etc., and pouring the decoction on the operator's hands. In all cases except the last lot of cigarette inoculations, the checks were inoculated last by rubbing the leaves of healthy plants with the wet fingers after washing.

The results seem to indicate definitely that occasional plugs of tobacco carry the mosaic virus, and possibly that certain brands are likely to be found to be rather consistent carriers of the disease, although others may carry it only rarely or possibly be free. Much more extensive work is of course necessary to determine these points. However, four of five plugs of Apple Suncured, three of four plugs of Reynolds' Natural Leaf, two of five plugs of Brown's Mule, and one of three plugs of Climax tested were found to give positive results; whereas of four plugs of Drummond tested, all gave negative results. Nineteen other plugs of miscellaneous brands gave only negative results.

The shredded chewing tobaccos to which sweetening is added appeared to be free from the virus and probably are put through a sterilizing process in manufacture. The granulated smoking tobaccos and cigarettes tested appear to be viruliferous, as positive results were obtained from all of them. Chesterfield cigarettes were used in a plant pathology student laboratory exercise and as a result of carefully controlled inoculations three plants became infected.

Of a total of 45 commercial brands of tobacco tested, infections were obtained from the following 16 brands:

Plug	Granulated Smoking	Cigarettes
Reynolds' Natural Leaf	Old Loyalty	Chesterfield
Apple Suncured	Five Brothers	Lucky Strike
Brown's Mule	Stud	Camel
Climax	Bull Durham	Herbert Tareyton
	Duke's Mixture	Piedmont
		Fatima
		Melachrino

SUMMARY

The results reported indicate that, in regions where cured tobacco is commonly chewed by tobacco growers, it may be an important source of mosaic infection, especially at weeding and pulling time. Commercial chewing tobaccos probably are not so important a source of mosaic infection, as far as tobacco is concerned, as the natural leaf, the results, except for four brands of plugs, being negative. Cigarettes and granulated smoking tobaccos also probably play a very minor part in the mosaic problem in commercial planting of tobacco. They are likely to be of importance, however, in connection with more limited plantings of susceptible plants such as tomatoes grown under glass or intensive culture in the field. Under these conditions the plants are handled from time to time in setting, pruning, training, and harvesting. Infection of a single plant under such conditions may lead to the spread of the disease through the house or field. Although it has not yet been conclusively demonstrated, it might be expected that the fingers of an inveterate cigarette smoker would become contaminated with the mosaic virus and thus transfer it to plants which were handled. Cigarettes are also an important consideration for the investigator who smokes cigarettes and works with susceptible plants. It it not at all improbable that many cases of sporadic mosaic infection occurring in cultures of tomatoes and tobacco in mosaic-free houses may have originated from fingers which have become contaminated while smoking cigarettes.

KENTUCKY AGRICULTURAL EXPERIMENT STATION,
LEXINGTON, KENTUCKY.

THE EFFECT OF A STRAIN OF TOBACCO MOSAIC ON THE YIELD AND QUALITY OF BURLEY TOBACCO[1]

W. D. Valleau and E. M. Johnson

In a test carried on the past season to determine the effect of mosaic on quality and yield of Burley tobacco, such striking results were obtained that they are being published, although they cover but a single season, in the hope that they may stimulate further investigation. Growers in Kentucky pay very little attention to mosaic when it appears in the fields in the usual amounts of a trace to 10 or 12 per cent after setting, taking it to be an abnormal condition of the plant beyond their control. Occasionally, when most of the plants are affected, it causes considerable concern. If the plants grow fairly normally, many growers believe the cured tobacco is not injured, as no sign of the mosaic pattern remains after curing. The question as to whether later infections, such as those occurring at topping or suckering stages, cause injury to the cured tobacco has hardly been given consideration. In this connection, Chapman (p. 81)[2] states: "The amount of damage done by late mild attacks when the plants are maturing, or appearing on the sucker growth after topping, is practically negligible and, so far as can be learned, does not in any way injure the commercial leaf."

Two distinct types of mosaic are commonly found in tobacco fields in Kentucky. One of these causes severe stunting, distortion and, in the field, burning of the leaves, together with a distinct mottling; the other type causes only a slight retardation of growth, the leaves are rarely distorted or rugose, and the mosaic pattern is not conspicuous. The first causes marked reduction in yield and quality if inoculations are made soon after setting. The other, the mild type, does not cause marked injury. The mild type of mosaic was used in the following experiment:

Thirteen rows of standup White Burley tobacco (strain 36–12) were set with a setter, June 9, 1926. The odd rows were left as buffer rows in an attempt to prevent, as much as possible, the spread of mosaic from row to row. The second and eighth rows were inoculated at setting time, or as soon as growth commenced if the first attempt was not successful. The fourth and tenth rows were inoculated at topping time, and the sixth and twelfth rows were not inoculated. The disease spread some during the summer, but care was used in selecting plants for final harvest, in the rows

[1] Published with the approval of the Director of the Kentucky Experiment Station.

[2] Chapman, G. H., Mosaic diseases of tobacco. Mass. Agr. Exp. Sta. Bul. 175. 1917.

TABLE 1.—*Effect of mild mosaic on the yield and quality of Burley tobacco at Lexington, Kentucky, in 1926*

Grade		Advance rates	Healthy uninoculated			Inoculated at topping			Inoculated at setting			
			Actual yield, 50 plants, gms.	Per cent	Advance per 1,000 lbs.	Actual yield, 50 plants, gms.	Per cent	Advance per 1,000 lbs.	Actual yield, 50 plants, gms.	Per cent	Advance per 1,000 lbs.	Advance per acre, yield 671 lbs.[a]
Flyings	A 6	$.07	533	13	$ 9.02	180	4	$ 3.06	429	16	$10.83	$ 7.27
Second trash	B 5	.13	773	19	24.30	561	14	17.71	.....	...	.....	.....
do	B 6	.09	.....	...	.....	279	7	6.09	.....	...	.....	.....
do	B 7	.05	.....	...	.....	313	8	3.80	755	27	13.61	9.13
Lugs	C 6	.10	598	14	14.46	515	12	12.50	.....	...	.....	.....
do	C 7	.06	.....	...	.....	.....	...	.....	333	12	7.21	4.84
Leaf	D 6	.09	727	18	15.83	340	8	7.43	.....	...	.....	.....
Leaf, green cast	DMI	.02	.....	...	.....	830	20	4.03	.....	...	.....	.....
Red	E 6	.07	1,250	30	21.16	.....	...	.....	.....	...	.....	.....
do	E 7	.04	.....	...	.....	703	17	6.83	983	35	14.18	9.51
Tips heavy	F 5	.04	254	6	2.46	398	10	3.86	273	10	3.94	2.64
Total			4,135	100	$87.23	4,119	100	$65.31	2,773	100	$49.77	$33.39
Per cent decrease in crop-value due to mosaic				0			25.1			43.1		61.7

[a] Assuming an acre yield of 1,000 pounds for the "healthy" and for the "inoculated at topping" plants, the yield per acre of the "inoculated at setting" plants would be 671 pounds.

inoculated at topping time, which appeared healthy up to the time of topping (August 11, 1926). The hands were washed in soap and water before topping the healthy rows. As a further precaution, these plants were examined nine days before cutting and only the plants with mosaic-free suckers were suckered, thus marking them for harvest. All plants in the other rows were suckered at the same time. Fifty plants were harvested from each of the three pairs of rows twenty-eight days after topping. The tobacco was all cured together, was stripped and sorted into hands, and was then taken to the Burley Cooperative Association warehouse where it was graded by the head grader of the Association and his assistant. The grade, advance paid by the Association, the actual yield in grams of each grade, the percentage of the total yield represented by each grade, and the advance for each grade on an acre basis, figuring the yield at 1,000 pounds per acre, are given in table 1.

The yield was reduced 33 per cent by infection at setting time, although not appreciably reduced by infection at topping time. The leaves of plants inoculated at setting time averaged about three or four inches shorter than the leaves of the other two lots, and were of lower quality. The leaves of plants inoculated at topping time fell into nine grades; whereas those from healthy plants fell into only six grades. This is an important factor, as it increases the difficulty of grading or, if the grading is not carefully done, reduces the value of the better grades because of admixtures of lower grades. The effect of mosaic seems to be evident even in the lower leaves (second trash and flyings) but is probably most marked in the quality of the leaves which expanded following inoculation.

In the leaves which expanded (grades D to F) there was a marked effect on color, 20.15 per cent of the crop falling into DMI which is "leaf" with a green cast. The E grade or red was greatly reduced in weight, and was of a lower grade than in the healthy plants, a portion of it evidently having fallen into the DMI grade. It seems certain that mosaic infection, even though not occurring until topping time, has a marked effect on the color of cured tobacco, causing it to be dark or to have a green cast.

The advance paid for the various grades of tobacco by the cooperative association, although not an accurate measure of its total value, may be used for comparison. A reduction of 33 per cent in yield due to inoculation at setting time resulted in a reduction of 43.1 per cent in value of a given weight of tobacco and 61.7 per cent reduction in value on an acre basis. Inoculation at topping time, although resulting in no appreciable reduction in yield, caused a reduction of 25.1 per cent in the value of the crop. This difference could hardly have been predicted from the appearance of the plants at cutting time. The result emphasizes the necessity of developing

cultural practices which will not only eliminate initial mosaic infection but will reduce the spread of the disease throughout the entire growth period of the plants.

It has been recognized for years that late inoculation, even in the upper portion of the plant, would result in the entire plant becoming viruliferous in a comparatively short time. That the presence of the virus in well-developed, apparently normal leaves has any effect upon them either while alive or during the curing process has not been generally recognized even by tobacco specialists, it being commonly believed that the chief loss caused by mosaic is due to stunting of the plant and to loss of quality in the mottled leaves.

Miss Eckerson[3] gives a possible basis for an understanding of the results reported in the present paper. After inoculation of a single leaf on a tomato plant, she found small flagellate organisms penetrating the chloroplasts of other leaves and partially destroying them, noting that later some cells were completely disorganized. If similar effects are found to be produced in mature tobacco leaves, it might be expected that during the curing process many of the partially disorganized chloroplasts, which have not lost their color completely, might not break down normally, but remain as they were, thus accounting for a darker color in some leaves and the distinctly green cast in others. The partial or complete destruction of other cell constituents following inoculation may also have some effect on the color of the cured product.

SUMMARY

1. Two types of mosaic occur commonly in the tobacco fields of Kentucky. One type produces severe stunting accompanied by distortion and a distinct mottling, together with a "burning" of the leaves; the other type causes only a slight retardation of growth, without an apparent quantitative or qualitative injury.

2. A study was made of the effect of the mild type of mosaic on the yield and quality of Burley tobacco, grown in the field in 1926.

3. There was no appreciable reduction in yield when the plants were inoculated at topping time; but infection at setting time resulted in a one-third yield reduction as compared with the check.

4. The leaves of plants inoculated at setting time averaged about three to four inches shorter and were of lower quality than the leaves of either the check or those inoculated at topping time, resulting in a reduction of 43.1 per cent in value of a given weight of tobacco and 61.7 per cent reduction on an acre basis.

[3] Eckerson, Sophia H. On organism of tomato mosaic. Bot. Gaz. 81: 204–209. 1926.

5. The leaves of plants inoculated at topping time fell into nine grades; whereas those from healthy plants fell into only six grades, thereby increasing the difficulty of grading.

6. Inoculation at topping time, although not reducing the yield, nevertheless resulted in a reduction of 25.1 per cent in the value of the crop, which difference could hardly have been predicted from the appearance of the plants at cutting time.

7. Possible disorganization of chloroplasts during the curing process, and the partial or complete destruction of other cell constituents following inoculations, may be responsible for the color of the cured product.

8. There is a real necessity for developing cultural practices which will not only prevent initial mosaic infection, but will reduce the spread of the disease throughout the entire growth period of the plants.

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THE BLACK WALNUT (*JUGLANS NIGRA* L.) AS A CAUSE OF THE DEATH OF APPLE TREES[1]

F. J. SCHNEIDERHAN

IN 1923 the writer observed an instance of incompatibility between a black walnut and apple trees in an orchard near Winchester, Virginia. The walnut, with a limb spread of about 40 feet, had apparently dwarfed two apple trees and killed six others in a circle around it. Digging up the roots of the injured apple trees revealed the fact that they were intermingled with walnut roots about 45 feet from the trunk of the walnut. This was the first instance that came to the writer's attention of injury resulting from close association of these two species of trees. In 1926, when a definite effort to find additional cases of this antagonistic relationship was made, we were informed by a farm hand employed for years on an old Virginia estate that he had been aware 30 years ago of the injurious effect of walnuts on apple trees. According to his version, it was rather generally known at that time that walnut trees injured or killed apple trees planted nearby.

The antagonism of certain species of *Juglans* to other plants is not a new observation. The notes of A. H. Gilbert reported by Jones and Morse[2] in Vermont indicate that the shrubby cinquefoil (*Potentilla fructicosa* L.) was killed in areas about the base of butternut trees (*Juglans cinerea* L.). This area extended beyond the limb spread of the butternut. Examination of the roots of the cinquefoil showed that whenever they came in contact with, or were in close proximity to, the butternut roots, they were killed. The death of the weed in this instance was always associated with close contact with the butternut roots. Where an outcropping of rock prevented the growth of the butternut roots, the cinquefoil grew normally.

Another instance of walnut antagonism to other species was noted by Mel. T. Cook,[3] who reported the wilting of tomato and potato plants growing in the immediate vicinity of the black walnut. He writes:

"Attention has been called from time to time to a number of cases of wilting of potato and tomato plants which was undoubtedly due to walnut (*Juglans nigra*) trees growing in the immediate vicinity. The plants show a decided wilting but do not lose

[1] Paper No. 71, from the department of botany and plant pathology, of the Virginia Agricultural Experiment Station.

[2] JONES, L. R., and W. J. MORSE. The shrubby cinquefoil as a weed. Ann. Rept. Vt. Agr. Exp. Sta. 16: 188–190. 1902–1903.

[3] COOK, MEL. T. Wilting caused by walnut trees. Phytopath. 11: 346. 1921.

their color or die, as in the case of plants that have been attacked by wilt-producing fungi or bacteria, or struck by lightning. The range of the wilting coincides very closely with the spread of the root system."

In an unpublished note, Fromme describes this type of injury to tomato plants as follows:

"On several occasions in Virginia, a wilting of tomato plants growing in proximity to black walnut (*Juglans nigra*) has been noted. The first observation was made near Amsterdam in 1916. Areas of wilted plants were seen in two separate fields of tomatoes, and each area centered on a walnut tree growing in the fence row. The rapid wilting of the plants suggested bacterial wilt (*B. solanacearum*), but examination failed to reveal this organism. On inquiry it was learned that the occurrence of this type of wilt in proximity to walnut trees was a matter of common observation among farmers in the locality."

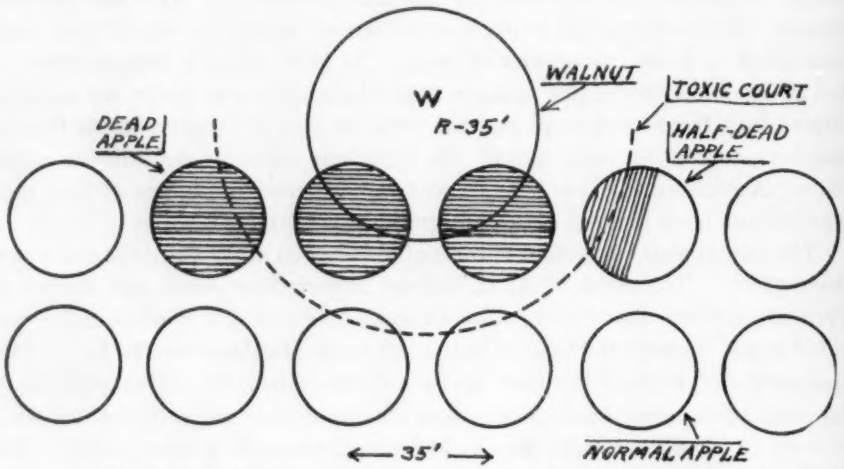

FIG. 1.—Walnut with a limb spread of 70 feet and a toxic court with a radius of 51 feet has caused the death of three York apple trees and the dwarfing of a fourth.

A more detailed study of the antagonism of walnut to other plants, together with the toxic constituent of the walnut, was made by Massey[4]. His conclusions are as follows, based on a study of the injury to alfalfa, tomatoes and potatoes:

"1.—Walnuts (*Juglans nigra* and *J. cinerea*) have an antagonistic action which causes a wilting and dying of certain plants such as alfalfa, tomato and potato.

"2.—Roots of the affected plant were always in close contact with walnut roots; the toxic substance is not generally distributed in the soil around the walnut trees, but localized in the vicinity of the walnut roots.

[4] MASSEY, A. B. Antagonism of the walnut (*Juglans nigra* L., and *J. cinerea* L.) in certain plant associations. Phytopath. 15: 773–784. 1925.

"3.—Walnut root bark contains a substance which is toxic to the roots of tomato plants grown in water culture.

"4.—It is likely that juglone, or some similar substance, is the toxic constituent of the walnut."

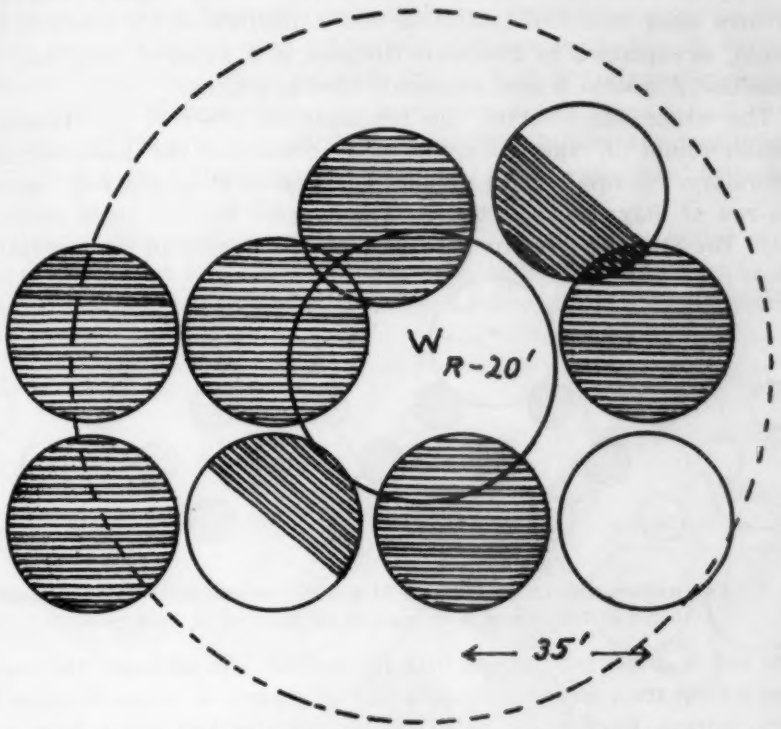

FIG. 2.—Walnut tree with a limb spread of 40 feet and a toxic court of 66 feet has caused the death of six York and Pippin trees and the dwarfing of two.

No evidence has been presented, so far as the writer knows, that species of *Juglans*, other than *J. nigra* and *J. cinerea*, are toxic to other plants, particularly the apple. There is, in fact, considerable evidence to the contrary with respect to species in common use as stocks for the Persian or English walnut. In a letter of November 2, 1926, to F. D. Fromme, Robert W. Hodgson, of the University of California, writes with reference to the injury in question:

"In my ten years of experience with the walnut in California, where we now have more than 100,000 acres, I have never seen any results of this kind. To be sure, our trees are not propagated on the eastern black walnut, *Juglans nigra*, but are either on the Persian seedling stock or one of the other of the two California black walnuts. Fully 50 per cent of our walnut acreage is intercropped, and practically all types of field and truck crops have been used successfully with no apparent evidence of injury. Thousands

of acres of tomatoes and potatoes are grown in young walnut orchards in Southern California. It is not at all uncommon to find apple trees in walnut orchards, and apparently doing very well.''

The two California black walnuts referred to are *Juglans hindsii*, the northern black walnut of California, and *J. californica*, the southern black walnut, as explained by Professor Hodgson in a letter of later date. He states that *J. hindsii* is most commonly used as a stock.

The writer has observed the non-injurious effect of the Persian or English walnut (*J. regia*) on apples on the grounds of the Winchester Field Laboratory. A row of these walnuts was planted about ten years ago next to a row of Stayman apple trees. The distance between these trees is 27 feet. The Stayman trees are growing normally in spite of the fact that the

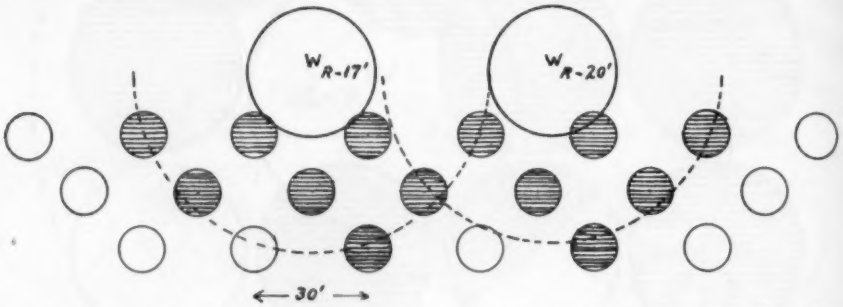

FIG. 3.—Two walnuts with limb spreads of 34 and 40 feet and toxic courts with radii of 46 and 45 feet, respectively, causing the death of 13 York trees.

roots and branches intermingle with the walnut. In addition, the roots of these walnut trees extend through a plat of ground in which Japanese barberry, spireae, English ivy, phlox, tulips, petunias, iris, castor bean, roses, and other flowers and shrubs have been planted. No injury of any kind has been observed during the past four years as a result of this association.

Our search for additional instances of walnut toxicity to apple trees was made in July and August, 1926. This work was incidental to our regular fruit disease investigations at Winchester. Only two days were given entirely to search for walnut injury to apples. In these two days we found seven instances along the country highways, and undoubtedly numerous additional cases could be found. Our purpose was merely to discover, if possible, additional cases of this type of injury without studying specific toxic products of the walnuts as causative factors. We found it to be a comparatively easy matter to discover instances of walnut injury while the foliage was still on the trees, a missing tree or an injured one being very noticeable at that time. The various measurements recorded later in this paper were made in January, 1927.[5]

[5] The writer is indebted to Dr. F. D. Fromme and Dr. S. A. Wingard, of the Virginia Experiment Station, for assistance in making measurements and for suggestions regarding the preparation of the diagrams. Acknowledgment of this assistance is hereby made.

The apple plantings in Frederick County, Virginia, are probably as extensive per unit area as in any part of the United States. The black walnut (*J. nigra*) is one of the commonest nut-producing trees in Virginia, and the wood of this tree has been used extensively in furniture manufacture since colonial days and is still used extensively in factories specializing entirely in walnut furniture. Thus we were able to find 18 instances of walnut injury, 13 of which were selected for description in this paper.

Judging by the ease with which we found 18 instances of walnut injury, we are of the opinion that the aggregate loss of apple trees caused by walnut toxicity is considerable in this state.

From the diagrammatic representation of the apple trees killed or injured by walnuts in the following pages, together with the photographs, the reader will be able to see that, with few exceptions, apple trees were either

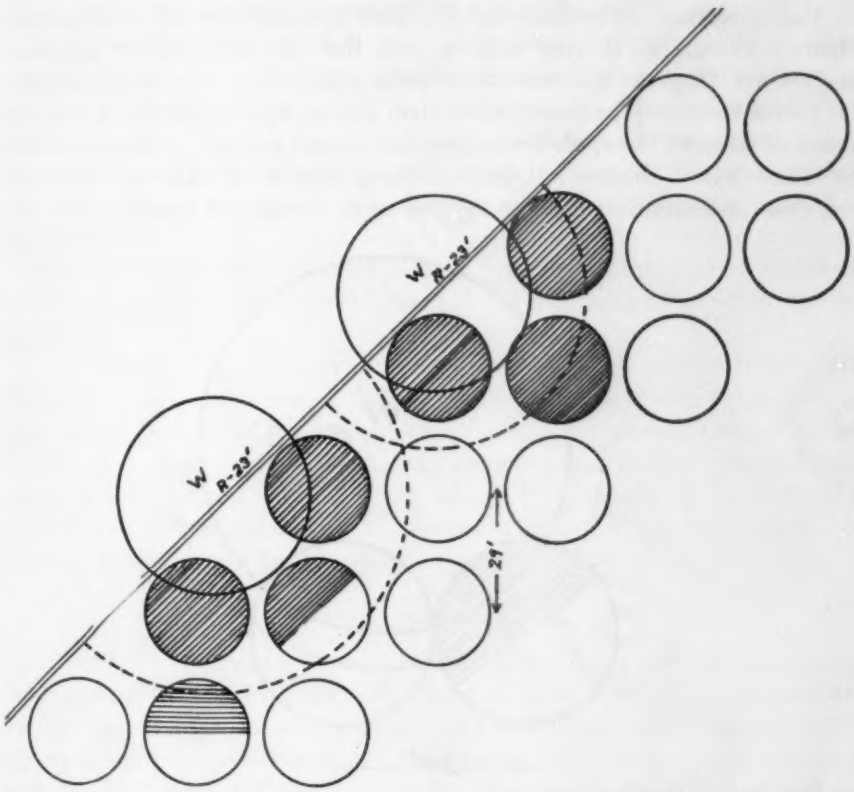

FIG. 4.—Two walnuts with limb spreads of 46 feet each and toxic courts with radii of 38 and 48 feet, causing the death of five York trees and the dwarfing of one tree inside and another outside of the assumed limits of the toxic courts.

killed completely or partly, or dwarfed when growing within the approximate limits of root spread—the toxic court surrounding the walnut. Usually this court is represented as a sector with an arc of 180 degrees because most of the instances of injury were found along fence lines bordering the orchards. In only one instance (Fig. 2) did we find a walnut growing within an orchard. Digging of roots to determine their extent and direction was not resorted to except in the case of the walnut tree in figure 2. In this instance we found that the walnut and apple roots intermingled, with unquestionable injury to the latter. The work of Massey indicates that there is considerable variability in the direction and extent of walnut root growth. The so-called "death line" of alfalfa plants surrounding a walnut indicates that the roots of the walnut extended from 33 to 64 feet from the trunk. Whenever uninjured apple trees were found within the limits of the toxic court, we assumed that the roots of the walnut had not developed in that direction. The fact that the average depth of soil in Frederick County (Virginia) is very shallow and that outcroppings of limestone shelves are frequent and numerous lends plausibility to this assumption. Of course there may be causes, other than walnut toxicity, of the injury and death of some of the apple trees described in this paper. Such causes may be mouse injury, shallow soil, root rot fungi, and other diseases. However, the close association of dead or injured apple trees with walnut trees, and

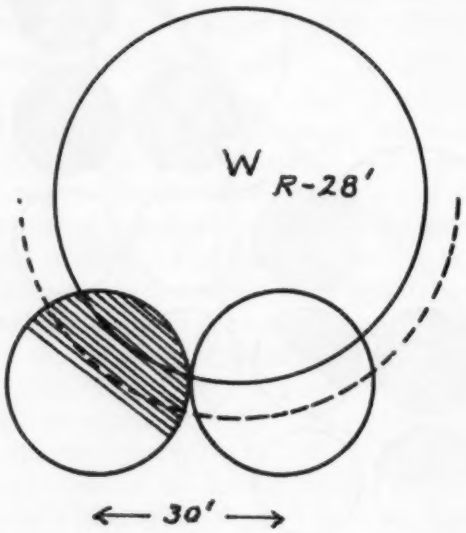

FIG. 5.—Walnut with limb spread of 56 feet and a toxic court with a radius of 36 feet, causing severe dwarfing of a Ben Davis and no apparent injury to a Stayman tree within the toxic court.

the regularity of this occurrence, lead us to conclude that walnut toxicity is the outstanding cause of death of apple trees in these specific cases.

An interesting observation requiring further study was made in regard to the comparatively small extent of walnut injury to trees of the Stayman variety. It appears that this variety either escaped injury in three instances or else possesses a varietal resistance to walnut toxicity not found in such varieties as the York, Ben Davis, and Pippin. Notable among these instances is the one represented in figure 5. In this instance, we found a Stayman tree growing normally at a distance of only 25 feet from a very large walnut; whereas a Ben Davis tree standing only 8 feet farther away was not only severely dwarfed, but all of the limbs on the side toward the walnut were dead. In another instance, we discovered two large Stayman trees uninjured at a distance of 43 feet from a large walnut, although in the same orchard row about six rods distant three Ben Davis trees were killed by a walnut growing at approximately the same distance.

These observations of the behavior of Stayman are too limited to make definite deductions. There is a possibility, nevertheless, that resistance to walnut toxicity by Stayman may be demonstrated. The explanation that presents itself is that this variety may have developed scion roots which may be resistant to the toxic constituent of the walnut roots. It is quite apparent that the ordinary crab stock on which our common varieties are grafted is not resistant.

The writer has used diagrams in describing five of the thirteen instances of walnut toxicity. The symbols used in the diagrams (Figs. 1–5) are explained here.

The largest circles with the letter W in them denote walnut trees. The letter R in these same circles followed by a figure indicates the radius, in feet, of the limb spread of the walnut. The fully shaded smaller circles denote dead apple trees. The partly shaded circles indicate seriously injured or dwarfed apple trees. The dotted lines, usually in the form of a semi-circumference, denote the approximate extent of the toxic court. In figure 1 these symbols are identified by a printed legend.

DISCUSSION OF FIGURES 1 TO 5

Figure 1. Typical injury of apples resulting from close proximity to a large walnut tree. The large limb spread of the walnut in this instance was 70 feet, and the approximate extent of the toxic court, as represented in the diagram, comprises a sector with a radius of 51 feet. Irregularity of walnut root development is indicated in this diagram, as the dwarfed tree is at exactly the same distance from the walnut as the dead trees. Figure 6 is a photograph of this instance of walnut toxicity. This protograph also shows

FIG. 6.—The toxic court of the walnut described in figure 1. Note the two small replants in the foreground, one of which is indicated by the flag.

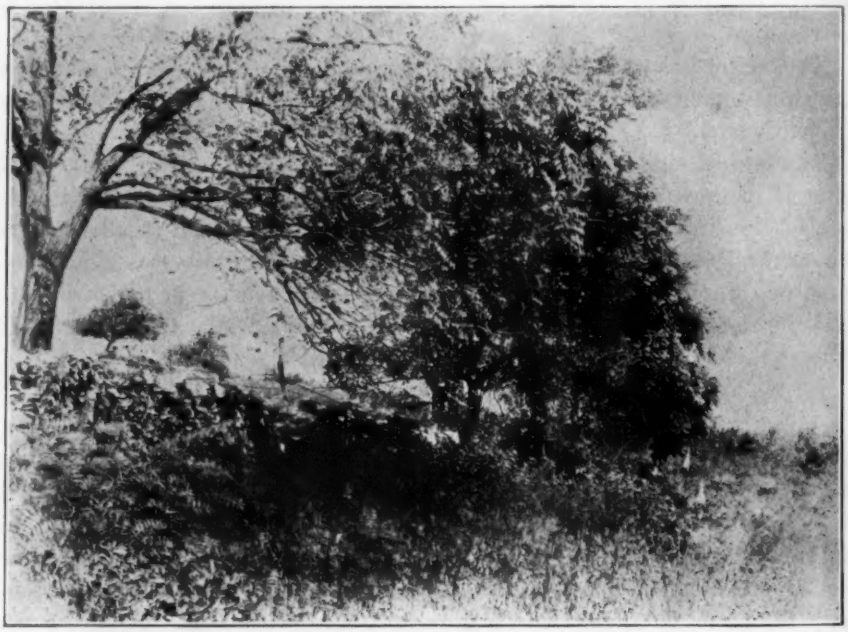

FIG. 7.—Two large walnuts which caused the death of 13 apple trees. The toxic courts of these walnuts are shown diagrammatically in figure 3. Some of the places occupied by the dead trees are marked by flags.

two small replants in the place of the dead trees. It is obvious that these replants will also be killed unless the walnut tree is removed.

Figure 2. This represents the first instance of walnut antagonism to apple trees noted in this study. After pointing out the injurious effect of this walnut to the owner of the orchard, the walnut was removed. This instance of incompatibility is noteworthy because it represents the largest number of apple trees killed by one walnut. Furthermore, we have here a comparatively small walnut with an extensive root system. The extent of the toxic court has been represented by a circle with a radius of 60 feet. The varieties are York and Pippin.

Figure 3. The loss of 13 trees was caused by two walnuts. In this instance, however, there were filler trees between the regular rows, which increased the number of tree deaths. Figure 7 shows a photograph which amplifies the diagram in figure 3.

Figure 4. Typical toxic courts are shown in a York orchard. The walnut trees in this instance are large, but the number of dead apple trees is comparatively smaller than in some other instances where walnut trees are smaller. Figure 8 is a photograph of this case of toxicity.

Figure 5. A very large walnut has severely dwarfed one side of a Ben Davis tree growing at a distance of 38 feet. A Stayman, on the contrary, growing at a distance of only 30 feet, was not injured. This is the smallest toxic court observed around so large a walnut tree.

EIGHT ADDITIONAL CASES OF WALNUT TOXICITY

No. 1.—In this instance a walnut with a limb spread of 50 feet and surrounded by a toxic court with a radius of 48 feet caused the death of two York trees within the toxic court and severely dwarfed another tree six feet outside of it. Two other trees outside the toxic court were also killed. To assign walnut toxicity as the cause of the death of the two last mentioned trees is questionable. However, the irregularity of root growth of walnut trees may account for this, but other factors may be involved. If the trees outside of the toxic court were killed by the walnut tree, they represent the maximum distance at which apple trees have been killed in this observation. The distance in this case was 80 feet.

No. 2.—In a well managed Ben Davis orchard the only missing trees were found to be within the toxic court of a walnut tree with a limb spread of 52 feet, the toxic court having a radius of 43 feet. As these are the only trees missing in the entire orchard and are found next to a walnut tree, there is little doubt that the walnut tree caused the death of the apple trees.

No. 3.—In the same orchard shown in figure 7, a walnut tree with a limb spread of 46 feet and a toxic court of 43 feet caused the death of three York

FIG. 8.—The large walnuts at the right of the picture caused this toxic court in which 5 trees were killed and 2 severely injured on the side toward the walnut trees. This instance of walnut toxicity is described diagrammatically in figure 4.

trees within the toxic court and the severe dwarfing of one tree standing 8 feet outside of the limit of the court.

No. 4.—A walnut tree with a limb spread of 48 feet and a toxic court of 36 feet caused severe dwarfing of two Ben Davis trees within the limit of the toxic court and of another tree seven feet outside of this limit. The dwarfing in all three instances was on the side of the tree toward the walnut.

No. 5.—Five Ben Davis trees were dead and a sixth severely dwarfed near a small walnut tree with a limb spread of only 46 feet and a toxic court with a radius of 66 feet. The dwarfed tree was outside of the toxic court.

No. 6.—A small walnut tree with a limb spread of 34 feet and a toxic court of 54 feet caused the death of three trees within the toxic court and the dwarfing of another tree outside of it. In this instance, four trees standing within the toxic court were uninjured.

No. 7.—A Stayman tree growing at a distance of 25 feet from a walnut was uninjured, whereas a Black Twig standing 43 feet from the walnut was dead. No other dead trees were found in this orchard. The limb spread of the walnut was 38 feet.

FIG. 9.—Walnut injury in one side of a Ben Davis tree. All of the branches on the side toward the walnut tree in the background have been killed. Figure 5 explains the toxic court of this walnut.

No. 8.—A large walnut with a limb spread of 52 feet apparently caused no injury to two Stayman trees 43 feet away. These Stayman trees were in the same row as the Ben Davis trees described as case No. 2. In view of the fact that the walnut tree is approximately of the same size and growing at the same distance from the Staymans as the walnut near the Ben Davis trees, it would seem to indicate varietal resistance of the Staymans to walnut toxicity.

SUMMARY

The data presented in the thirteen instances of walnut toxicity indicate that a total of 16 walnut trees have apparently caused the death of 48 apple trees of different varieties, or an average of 3 apple trees per walnut. A total of 14 apple trees were either dwarfed or injured on one side by these same walnuts; the average distance of the dead trees from the walnuts is 39 feet; the average distance of the dwarfed and partly injured trees is 47 feet. The maximum distance at which an apple tree was probably killed by walnut toxicity is 80 feet. The average limit of the toxic court is a sector drawn with a radius of 50 feet from the walnut as a center. The maximum

extent of this toxic court is such a sector with a radius of 70 feet. The average limb spread of walnuts in this study was 46 feet, and the circumference of the walnut trunks, 3 feet from the ground, was 5 feet and 4 inches. The ratio of the trunk circumference to the limb spread in feet is as 1 to 8.4. The average limb spread of apples examined in this survey is 13 feet. There is no correlation between the extent of walnut limb spread and the toxic court. This is shown very strikingly by the fact that one walnut tree with a limb spread of 56 feet has a toxic court of approximately 36 feet; whereas another walnut tree, with a limb spread of only 34 feet, has a toxic court of approximately 66 feet.

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VARIETAL SUSCEPTIBILITY OF ADA RED AND CERTAIN OTHER APPLE VARIETIES TO CEDAR RUST, WITH SPECIAL REFERENCE TO TWIG INFECTIONS

V. H. YOUNG

Varietal differences in the susceptibility of certain kinds of apples to leaf and fruit infection with cedar rust have been commonly noted, and certain writers have further observed that some varieties are also susceptible to twig infection. Reed and Crabill (4) find that on the variety Smith's

FIG. 1.—Cedar rust lesions on apple nursery stock of the variety Ada Red. ×1½.

Cider the twigs are frequently seriously diseased by cedar rust. Giddings (1) pictures mature aecia of the cedar rust pathogen on wild crab apple twigs and, more recently, Hopkins (2) reports marked susceptibility to twig infection in the Yellow Bellflower. Jones and Bartholomew (3) also report twig infection on the wild crab in Wisconsin, and Weimer (5) reports twig infection of apples with cedar rust. So far as the writer knows, the relation of twig infection to young nursery stock has not been considered.

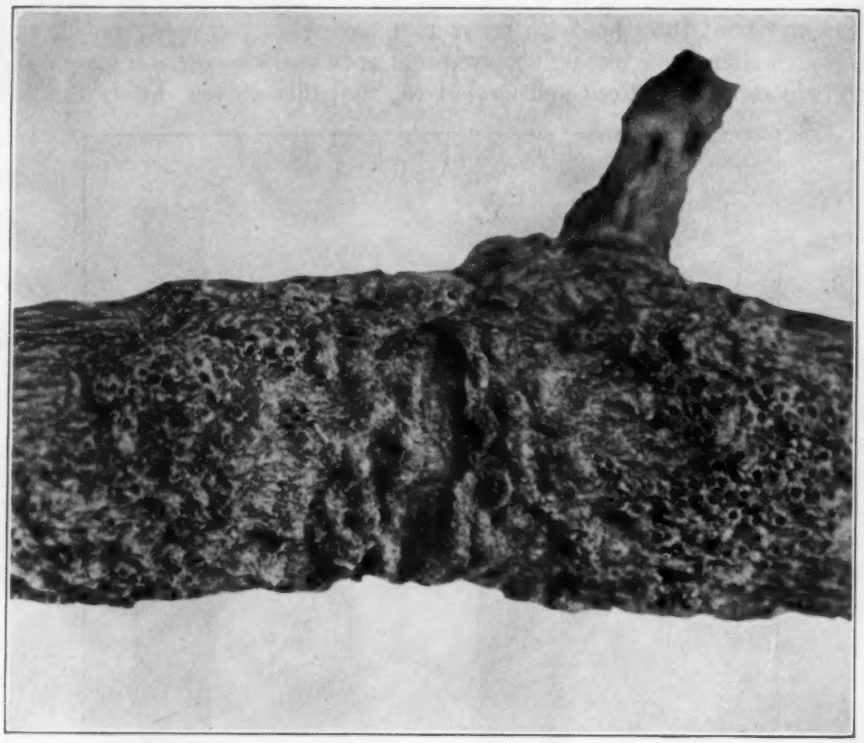

FIG. 2.—Mature aecia on cedar rust lesion on apple nursery stock. ×4.

During the fall of 1926, Mr. Sam E. Poe, of the staff of the Arkansas State Plant Board, called the writer's attention to a serious attack of cedar rust on the wood of one-year-old whips of the variety Ada Red. In one nursery in Benton County, Arkansas, in a block of several thousand one-year whips of this variety, more than 9 per cent of the trees were badly affected with cedar rust on the main stem. In another nursery, a few miles away, about 7 per cent of the trees in a block of the same variety were affected. Blocks of other standard varieties such as Delicious, Winesap, and Arkansas Black in the same nurseries were not affected with twig infec-

tions. In practically all cases the injury occurred about a foot above the crown and was in the form of a deep-seated canker, which in many cases had completely girdled the stem (Fig. 1). Many of the trees were so severely affected that they broke off when slightly bent, and early in the fall a large number of them broke at the point of infection from the weight of a light snowfall. Such trees in many cases were found to be dead above the point of infection, and in all cases the injury was so severe as to render the trees worthless. No shortening and thickening of the growth, such as is noted by Weimer (5) in twig infections, was seen.

Mature aecia were noted by the writer on many of the cankers, and upon microscopic examination the peridial cells and the contained spores were found to agree closely in their measurements and appearance with those of *Gymnosporangium juniperi-virginianae* Schw. (Fig. 2). Twig infection on mature trees of the variety Ada Red has been commonly noted in northwest Arkansas by the writer and by others, but apparently little serious injury results in most cases. This variety, in common with the Jonathan, Golden Delicious, and certain other varieties, often suffers severely from cedar rust infections on the leaves, and to a less extent from infections on the fruit. Infection of the wood of one-year whips of Ada Red is apparently a much more serious matter than twig infection of older trees, and results in a total loss of the year's growth. As was noted by Hopkins (2), wood infections appear to originate near buds rather than on the internodes, and from the position of most of the cankers a few inches from the crown it is inferred that only the apical portion of a growing twig is susceptible to infection.

Severe infection on the wood of nursery stock has been noted in one case on the Golden Delicious, and twig infections on older trees are fairly common in northwest Arkansas on the varieties Jonathan, Ada Red, Benoni, Duchess, and Golden Delicious. It is worthy of note that the leaves of all these varieties, with the possible exception of Benoni, are also extremely susceptible to cedar rust.

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LITERATURE CITED

1. GIDDINGS, N. J. Infection and immunity in apple rust. West Va. Agr. Exp. Sta. Bul. 170. 1918.
2. HOPKINS, E. F. Varietal susceptibility of the yellow bellflower apple to cedar rust. Phytopath. 12: 190–192. 1922.
3. JONES, L. R., and E. T. BARTHOLOMEW. Apple rust and its control in Wisconsin. Wisc. Agr. Exp. Sta. Bul. 257. 1915.
4. REED, HOWARD S., and C. H. CRABILL. The cedar rust disease of apples caused by *Gymnosporangium juniperi-virginianae* Schw. Va. Agr. Exp. Sta. Tech. Bul. 9. 1915.
5. WEIMER, JAMES LEROY. Three cedar rust fungi: their life histories and the diseases they produce. N. Y. (Cornell) Agr. Exp. Sta. Bul. 390. 1917.

DOTHICHIZA POPULEA AND ITS MODE OF INFECTION

GEORGE G. HEDGCOCK

Dothichiza populea Sacc. and Briard causes a serious canker disease of some species of *Populus,* especially in the Northern and Northeastern United States and Southeastern Canada (2, 3, 4). This fungus, apparently introduced from Europe where it was first described in 1884 [according to Delacroix (1)], attacks most severely the Lombardy and Norway poplars (*Populus nigra italica* and *P. eugenei*).

The life history of *Dothichiza populea* is not fully known. The writer (3), during his earlier studies of this disease, observed that cankers are almost invariably formed at the nodes, and frequently around the bases of immature dead twigs. Detmers (2), in 1923, stated as follows, regarding its mode of infection: "Infection takes place at the nodes. The discolored area may be immediately below the leaf scar, in the axil of the bud, or on either side of the bud in proximity to the stipular scars. The bud itself is not attacked. Cankers form at any node on two-year-old wood." Her observations as to the location of the infection the second year are correct, but convey the impression that the disease gains entrance through the leaf and stipular scars, and axils of the buds.

The writer in 1922 carried out two experiments to ascertain the manner of infection. Young healthy trees of *Populus* and *Salix* without bark lesions or cankers were grown in a bed near the greenhouses of the U. S. Department of Agriculture at Washington, D. C. In one experiment the following trees were inoculated: 3 *Populus alba* L., 10 *P. alba* L. var. *pyramidalis* Bunge., 3 *P. deltoides* Marsh, 5 *P. eugenei* Simon-Louis, 7 *P. nigra* L. var. *italica* Du Roi, 10 *Populus* sp., and 3 *Salix* sp. To obtain spores for inoculation, the lower ends of twigs of *Populus nigra italica* infected by *Dothichiza populea* were placed in water under a bell jar until the spore tendrils exuded from the pycnidia. On May 13, 1922, the tendrils were removed and dissolved in sterile tap water and the spore suspension was applied with a sprinkling can to the leaves, twigs, and limbs of the trees mentioned above.

By August 1, many of the inoculated leaves on the species of *Populus* that later became cankered had become discolored with gray to black or brown irregular leaf spots, which gradually increased in size. The dead areas later extended to the petioles and darkened them. By September 15 some of the leaves and petioles were killed, and by October 15 the discolora-

tion had progressively spread from the leaf blades to the petioles and from them to a number of the smaller greener and more succulent twigs, which gradually blackened, shriveled, and died. The following spring, small discolored areas or cankers appeared in the bark of the trunks at the bases of the dead twigs, and in three instances at the bases of dead leaves at the nodes. Twig infections were noted on the following trees: 5 *Populus nigra italica*, 1 *P. deltoides*, 3 *P. eugenei*, and 4 *Populus* sp. All the infected trees of these species bore infected leaves. Cankers with fruiting tendrils of *Dothichiza populea* appeared at the bases of diseased twigs in the spring of 1923 on 5 *Populus nigra italica*, 2 *P. eugenei*, and 2 *Populus* sp. Control trees, one of each species, remained free from infection.

Dothichiza populea was reisolated from the tissues of the freshly diseased leaves, petioles, twigs, and cankers previously described, after sterilizing their surfaces with a solution of mercuric chloride in distilled water, 1 to 1,000. The fungus was obtained from 5 out of 6 leaf cultures, 5 out of 6 petiole cultures, and 6 out of 6 twig cultures.

On May 12, 1922, a number of other trees were inoculated by inserting spore horns obtained as in the other experiment in wounds made in the bark by means of a sterile scalpel. After inoculation the wounds were completely covered with a wrapping of dry, sterile absorbent cotton. The following trees were inoculated: 4 *Populus alba*, 4 *P. alba pyramidalis*, 4 *P. deltoides*, 4 *P. eugenei*, 7 *P. nigra italica* and 4 *Salix* sp. For controls, one tree of each species was similarly treated, but no spores were inserted in the wounds. None of the controls became infected. Of the inoculated trees, 3 *Populus nigra italica* and 1 *P. eugenei* were infected, and by September 15 small cankers appeared around the wounds. The cankers developed rapidly and produced the spores of the fungus by April, 1923. The tops of all four trees had also died above the cankers.

These experiments, though rather crude and carried out in the open under natural conditions, are offered as evidence that *Dothichiza populea* may infect poplar trees by progressive infection from the leaves through the petioles to the twigs and limbs, and also by direct entrance through wounds. They do not disprove, however, that the fungus may enter also through leaf and stipule scars and bud axils. Under natural conditions this fungus produces a large crop of viable spores in the spring about the time the poplar leaves are expanding. Repeated attempts by the writer to secure cultures of the fungus from infected specimens collected in autumn indicate that it produces few if any spores at that time. For this reason infections probably do not occur in late autumn after leaf scars have formed. Infections probably occur at the time the spores are produced, or soon thereafter.

It is possible that the entrance of the fungus through the leaves in springtime might be prevented, especially in nurseries, by spraying the trees with bordeaux or other fungicides about the time the leaves are expanding, and again after each heavy rain for at least one month thereafter. The writer has had no opportunity to test the effect of spraying and offers the suggestion to others who may have an opportunity to do so.

Publication of the results given here has been delayed in the vain hope that the experiments might be repeated with more refined technique, but they are now offered for what they may be worth to other investigators of the subject.

Since the first account of the occurrence of the disease caused by *Dothichiza populea* in the United States (3), the known range of the fungus has been greatly extended. Much of this extension has very probably resulted from the shipment of diseased nursery stock. The fungus has been collected from Maine southward to Virginia and westward to Minnesota, Nebraska, and New Mexico, on the following species of *Populus*: *P. brevifolia, P. deltoides, P. deltoides canadensis, P. eugenei, P. nigra italica, P. petrowskiana, P. simonis,* and *P. simonis fastigiata.*

SUMMARY

Experiments carried out under natural conditions indicate that *Dothichiza populea* Sacc. and Briard may infect poplar trees by progressive infection from the leaves through the petioles to the twigs and limbs, and also by direct entrance through wounds.

Spraying with bordeaux is suggested as a probable means of preventing the fungus from entering through the leaves in the springtime.

Since the first report of the disease in North America, the known range of this fungus has been greatly extended in Northern and Northeastern United States and Southeastern Canada, probably through shipment of diseased nursery stock.

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LITERATURE CITED

1. DELACROIX, G. Sur le parasitisme de *Dothichiza populea* Sacc. et Briard sur diverses espéces de peupliers. Bul. Soc. Mycol. France **19**: 353–355. 1903.
2. DETMERS, F. Dothichiza canker on Norway poplar. Phytopath. **13**: 245–247. 1923.
3. HEDGCOCK, G. G., and N. R. HUNT. *Dothichiza populea* in the United States. Mycologia **8**: 300–308. 1916.
4. MOSS, E. H. Observations on two poplar cankers in Ontario. Phytopath. **12**: 425–427. 1922.

THE NEMATODE DISEASE OF SWEET POTATOES[1]

R. F. Poole and Robert Schmidt

It has been known for some time that the sweet potato, *Ipomoea batatas*, is susceptible to the eel worm disease, *Heterodera radicicola* (Greaf) Muller (1, 2), but only recently has definite information been published regarding the susceptibility and resistance of sweet potato varieties (5). Nematode root knot is a major disease of many crops throughout the South and in greenhouses in many parts of the North. Its importance is further emphasized because the worm not only injures the root system, resulting in malformed roots and stunted plants, but it is accused of opening the tissues of roots and stems so that they become susceptible to fungi that cause the severe root rot and wilt diseases.

The 1926 season was an extremely favorable one for nematode infestation of plants in North Carolina. Cotton, soybeans, cowpeas, okra, beets, parsley, tomatoes, tobacco, carrots, lettuce, and celery are a few of the crops that were badly diseased in the field. The severity of the infestation should warrant an immediate presentation of any information on varietal resistance.

PLAN OF EXPERIMENT

The experiment had its origin as a varietal test for adaptability and quality of sweet potatoes conducted at the Coastal Plain Station, Willard, North Carolina, by the Horticultural Department. The infested soil at the station is of the sandy nature that is characteristic of the Atlantic coast from New Jersey to Florida. The test plots were sown in winter oats which were cut and plowed under about May 1. The soil was then prepared for sweet potatoes. A high grade fertilizer was applied in the ridge at the rate of 600 pounds an acre. On June 8, one week later, the plants were set out 12 inches apart in 3.5- foot rows. The following varieties were used: Red Jersey, Yellow Jersey, Big Stem Jersey, Porto Rico, Triumph, Southern Queen, Norton Yam, Nancy Hall, Yellow Yam, Red Bermuda, and Pumpkin Yam. Related strains of these varieties used were Vineless Yellow Jersey, Improved Big Stem Jersey, Old Long Red, Early Carolina, General Grant, Myers Early, Dixie Yam, Miles Yam, White Yam, and New Gem.

[1] Published with the approval of the Director of the North Carolina Experiment Station as paper Number 19 of the Journal Series.

NATURE OF NEMATODE INFESTATION

On some varieties, the stems, roots, and potatoes were infected. Knots were very prevalent on the small roots (Plate XIX, A), where the sizes varied from that of a pin point to three millimeters in diameter. The knots on the small potatoes (Plate XIX, B) were also large and protruded prominently. In only a few large potatoes out of thousands examined were the knots well developed and raised above the surface of the potato skin (Plate XIX, C, and Plate XX, A), for in most instances the roots were badly malformed, with deeply indented, rough scabs (Plate XIX, C and D). Even the larger potatoes were affected severely, there being uniform infection over

FIG. 1. Prominent scab-like infected areas on Yellow Jersey variety.

the entire surface (Plate XX, C). The indenting of circular areas around the potato roots (Plate XX, A) was a constant symptom on the severely infected varieties, where newly developed sprouts had been attacked and destroyed. The female worms were abundant in the knots on roots and on small and large potatoes (Plate XX, B, D) being most numerous along the margins of the large physiological cracks, both on small and large potatoes. They apparently were first attracted to these broken tissues that could be entered more readily than healthy parts. In some cases the points of entrance into the potato centered around the new sprouts and the roots that developed on the sides of the potatoes. Infection was most severe in

the cracked or broken tissues. The females contained an abundance of eggs, some of them with nearly mature worms. The males were not abundant at harvest. The few infected areas on the Jersey varieties (Fig. 1) were distinctly scab-like ones in which the cortical cells were broken and split open.

RELATION OF VARIETIES TO INFECTION

When examined early in October, the vines on severely infected varieties were not well developd. At that time a decision was made to obtain data on infection at harvest, which was October 21. It so happened that the varieties were alternated and repeated in such a satisfactory way that uniform infestation of the soil on the test plot was made certain.

Some relations of varieties to infection, yield under infected conditions, and remarks are given in table 1. Of the Jersey and Porto Rico varieties

TABLE 1.—*The relative susceptibility of important commercial sweet potatoes to nematode disease*

Variety	Infection (per cent)	Yield (bu. per acre)	Remarks
Red Jersey	0.8	245.0	No infection of roots.
Yellow Jersey	1.0	228.0	Very slight infection, confined mostly to ends of small roots.
Big Stem Jersey	1.2	238.0	Very slight infection, confined mostly to ends of small roots.
Porto Rico	1.2	237.0	No infection of roots, but slight infection of cracked potatoes only.
Triumph	5.8	120.5	Very slight infection of roots.
Southern Queen	35.5	107.3	Uniform but slight infection of roots.
Norton Yam	85.0	71.1	All roots badly diseased.
Nancy Hall	100.0	81.7	All roots and potatoes severely diseased, roots badly broken—potatoes severely marked.
Yellow Yam	100.0	28.1	All roots and potatoes severely diseased, roots badly decayed and broken—potatoes severely marked.
Red Bermuda	100.0	26.6	All roots and potatoes severely diseased, roots broken.
Pumpkin Yam	100.0	32.5	All roots and potatoes severely diseased, roots broken.

there was only an occasional knot on the roots and a few blister-like areas on the potatoes. The infection on these varieties was so insignificant as to be of no economic importance. On the roots of Triumph plants there were many knots, but only slight infection of potatoes. The roots of the Southern Queen showed minor infection, but the potatoes were irregularly, although severely, marked. Weimer and Harter (5) found no infection on this variety in tests made in California. They also reported no infection on the Red Jersey, which was only slightly attacked in these tests. Otherwise the results given in this paper are in agreement with those they obtained. Infection was very severe on Norton Yam, Nancy Hall, Yellow Yam, Red Bermuda, and Pumpkin Yam. The potatoes were so severely marked that it was impossible to make accurate counts of recently infected areas (Plate XX, C). The New Gem and Dixie Yam, which are strains closely related to the Porto Rico variety were only slightly infected. There was only slight infection also of the Jersey varietal strains, Improved Big Stem Jersey, Old Long Red, and Early Carolina; the varieties General Grant and Myers Early, which are related to Nancy Hall, were severely diseased. White Yam and Miles Yam, which are closely related to Southern Queen, had slightly infected roots and severely infected potatoes.

The severely infected varieties showed a marked reduction in yield compared with that of the more resistant varieties. The actual loss due to nematodes is suggested in the yields, since the Nancy Hall, Southern Queen and Yellow Yam normally yield as well as the Porto Rico. In most well fertilized soils the Southern Queen will yield better than the Porto Rico and Jersey varieties. The Porto Rico yield of 153.3 bushels an acre more than the Nancy Hall is largely owing to nematode infection of the latter. On the other hand, the Pumpkin Yam and the Red Bermuda may yield poorly on any soil, and their low yield in this test may be partly due to other factors, as it is well known that some of the varieties do not yield well on all soils (3). However, it may be safely stated that the low yields in these tests were due in great part to nematode infection.

DISCUSSION

It is probably most interesting to note here that varieties both susceptible and field-immune to wilt-producing species of *Fusarium*, were attacked by nematodes. The Nancy Hall variety is not only very susceptible to nematodes but is one of the most susceptible to wilt or stem rot. The Yellow Yam, which is immune from wilt or stem rot, was severely attacked by nematodes. The only wilt resistant variety that showed any worth while resistance to the nematode disease was the Triumph, but it is not a market favorite and would not be grown extensively for that reason. This brings

about an extremely complex situation in which the wilt- or stem-rot resistant varieties can not be used safely on soils infested with nematodes and with the causal Fusaria of wilt. Where such infections occur it will be interesting to revert to the skill of cultural methods, since they have been used successfully in reducing losses and maintaining normal yields.

Close planting and the use of two or three plants in hills formerly set with one has not only maintained a normal stand but produced a satisfactory yield (4). This is still the real hope of fighting the stem-rot disease on most infested soils, and should receive further attention in the South where the nematodes are likely to cause severe losses of the favorite varieties: Nancy Hall, Southern Queen, Yellow Yam, and Norton or Dooley Yam.

Porto Rico, the favorite variety in the South, is very susceptible to stem rot, and since it normally produces many "jumbos" (a term applied to potatoes larger than the market grades), could be set as close as 8 to 12 inches without reducing the yield of marketable potatoes. Under most conditions this could be counted on to maintain a normal stand and give a satisfactory yield on severely infested soils. The close plantings would certainly prevent the formation of large potatoes, and aid somewhat in satisfying market demands.

The Yellow Jersey varieties are very susceptible to stem rot or wilt but resistant to nematodes. Furthermore, the big commercial plantings of these varieties are north of the areas where the nematode is of economic importance in the field.

SUMMARY

The nematode or eel worm disease of sweet potatoes is the cause of heavy losses in the eastern part of North Carolina, especially on sandy soils. Cotton, soybeans, cowpeas, okra, beets, parsley, tomatoes, tobacco, carrots, lettuce, and celery are a few of the crops that were badly diseased in the field in 1926.

The popular commercial and other strains of sweet potato varieties which were tested on the infested soils showed wide variation in resistance and susceptibility. The Porto Rico and Jersey varieties were very resistant to the nematode, while all other important varieties, Norton Yam, Yellow Yam, Southern Queen, Red Bermuda, and Nancy Hall, were very susceptible.

Root infection of resistant varieties was very slight, there being only a few definite blister to scab-like scars on the potato. On the susceptible varieties the symptoms were so-called knots and scabs on stems, roots, and potatoes. On the susceptible varieties malformation was produced; scabby areas, followed by pit-like rots, were prominent; but raised pustule-like areas were not very abundant on potatoes.

Infestation was very prominent on potatoes that were cracked sometime during the season by physiological changes. The female worms were abundant in these areas and were full of eggs, which contained nearly mature worms.

Varieties susceptible to Fusarium wilts are resistant to the nematode, and vice versa. For instance, it would not be safe to use the Southern Queen, Yellow Yam, and Red Bermuda varieties, which are resistant to stem rot, in nematode-infested soils. Where both parasites are likely to be abundant, it is suggested that the Porto Rico and Jersey variety strains be planted, because of their resistance to nematodes. For the control of stem rot a sufficiently large number of plants should be planted per acre to insure a satisfactory stand after the period of heavy infection. This is done by setting plants 8 to 12 inches apart in the row, or 2 to 3 plants in 18- to 24-inch hills.

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LITERATURE CITED

1. BESSEY, E. A. Root knot and its control. U. S. Dept. Agr. Bur. Plant Ind. Bul. 217. 1911.
2. ELIOTT, J. A. Nematode injury to sweet potatoes. Phytopath. **8**: 169. 1918.
3. POOLE, R. F. The stem rot of sweet potatoes. N. J. Agr. Exp. Sta. Bul. 401. 1924.
4. ————————. Cultural methods for reducing sweet potato losses caused by stem rot. N. J. Agr. Exp. Sta. Bul. 433. 1926.
5. WEIMER, J. L. and L. L. HARTER. Varietal resistance of sweet potatoes to nematodes, *Heterodera radicicola* (Greaf.) Muller, in California. Phytopath. **15**: 423–426. 1926.

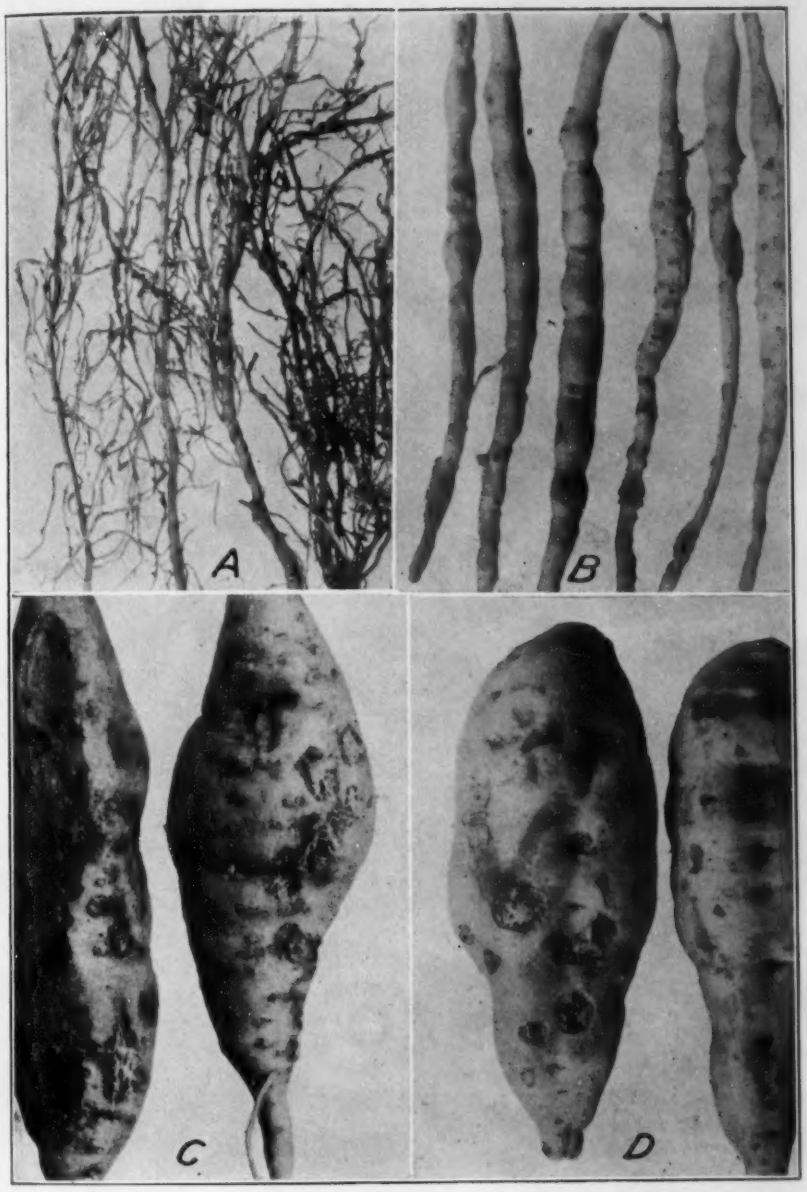
DESCRIPTION OF PLATES

PLATE XIX

A. Malformations or knots on small roots of Nancy Hall sweet potato.
B. Small potatoes badly diseased, and malformed.
C. Coarse scabby formation on surface; most severe on potatoes physiologically cracked.
D. Scab and pit areas on sprouting points.

PLATE XX

A. Scab areas extending around the potato.
B. Small potatoes and roots, in which female nematodes were abundant.
C. Large potato severely attacked by worms.
D. Large potatoes, in which female nematodes were numerous; greatest infection on physiologically cracked areas.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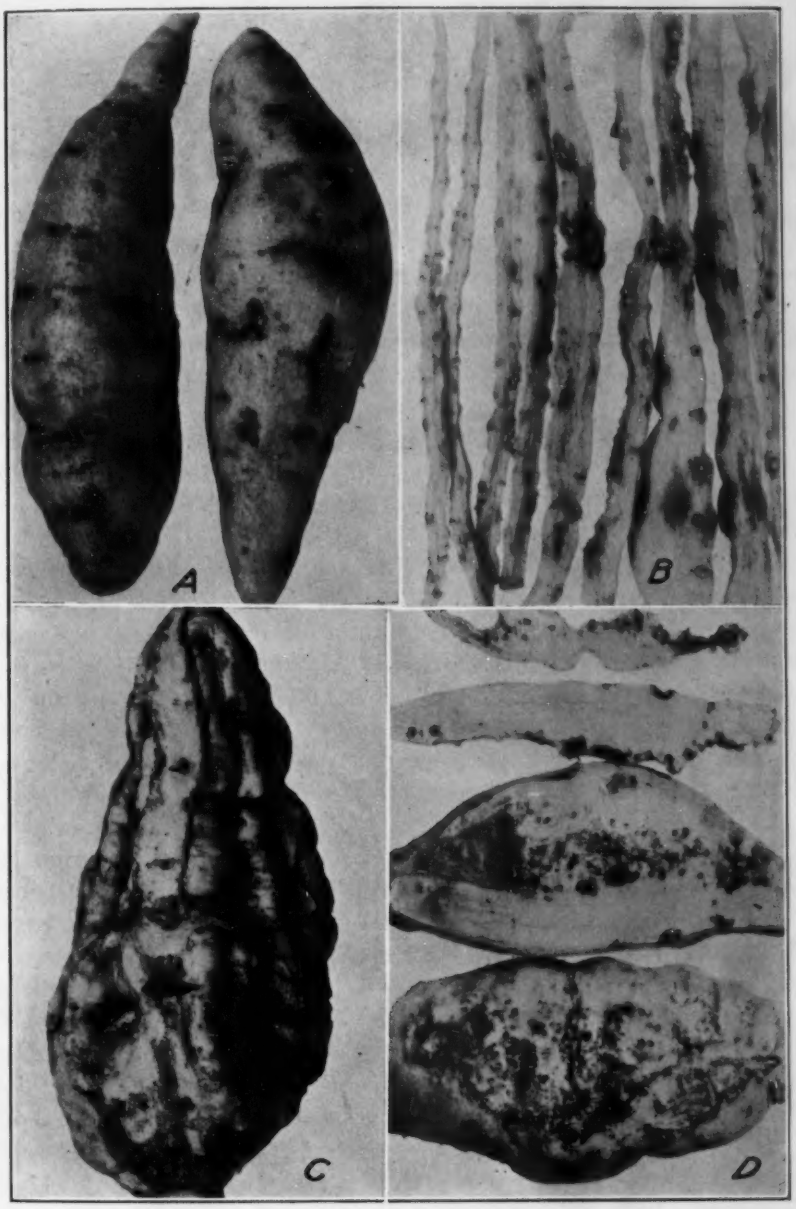
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NET NECROSIS OF THE POTATO

A. H. GILBERT

Some confusion appears to exist in the literature in regard to the terminology of potato tuber necroses. This is recently illustrated in an article entitled *Net Necrosis of the Potato* by D. Atanasoff (1). Atanasoff, in this article seems to have arrived at quite different conclusions from those reported in the United States by the present writer and others, and appears to have taken the term "net necrosis" from its connotation here and applied it to certain tuber and vine symptoms occurring in Holland, which symptoms, as described, are quite distinct from those associated with net necrosis in this country.

There are evidently several types of necrosis in potato tubers, both in Europe and in America. These include tuber discolorations variously known as brown fleck, internal brown spot, internal rust spot, sprain, streak, and net necrosis. The divergent use of these terms by various investigators, and others, indicates that there is a considerable lack of uniformity in their application. It appears, however, that there are some features more or less common to a group of these diseases and on the basis of which they may be distinguished from net necrosis. First, they affect various internal areas of the tuber but not specifically the phloem of the vascular tissues; second, the necrotic conditions are not progressive during storage as in the case of net necrosis; and last, these necroses are not transmitted by the tuber with the result that there are any characteristic observable effects upon the plants grown from affected tubers.

In noting the several necrotic conditions that may be observed in potato tubers, it should not be forgotten that frost necrosis, as described by Jones, Miller and Bailey (11), produces a browning and blackening of the vascular region and phloem strands which, in at least one of the several types, closely resembles net necrosis.

The writer has recently undertaken a study of net necrosis, of the phloem-necrosis type, as it occurs in tubers of the Green Mountain variety, and although the investigation is still under way and quite incomplete, it has seemed justifiable to make some preliminary statements in connection with general comments on the paper by Atanasoff (1).

In the first place, the point to be emphasized in opposition to the view of Atanasoff is that the net necrosis first noted and described by Orton (5) is the net necrosis which has more recently been extensively investigated by

Schultz and Folsom (8, 9, 10) and found to be associated intimately with leafroll. Results secured by the writer (2), including observations covering a period of several years, fully agree with those of Schultz and Folsom (8, p. 78), who state that "net necrosis is apparently a leafroll symptom, being a discoloration which results from tuber phloem necrosis and which appears more often as conditions of variety, recency of infection, and weight of tuber are more favorable. It develops in the dormant tubers without relation to differences in the storage temperature."

Kasai (3), reports from Japan a similar condition of tuber necrosis which was correlated with leafroll. Positive results in the way of transmission were secured by him by means of tuber grafts and also with juice inoculations. In a certain lot of leafroll tubers used for grafting, he reported flesh discolorations in twelve tubers. Kasai (3, p. 52), writes as follows in regard to the necrosis he described: "This, in all probability, is identical with the net necrosis of the tubers described by Schultz and Folsom (1921)."

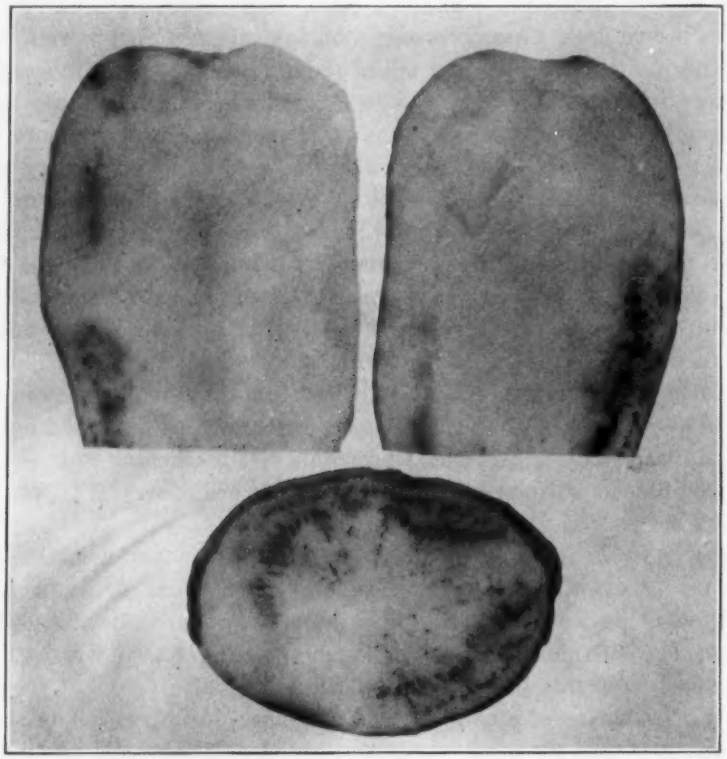

FIG. 1. Cross and longitudinal sections of a Green Mountain tuber with well marked net necrosis.

Net necrosis of the tuber as the writer has studied it may be described as a degeneration, browning, and ultimate dying of the cells of the phloem strands and phloem parenchyma, both outside and inside the vascular ring of the tuber. This degeneration and browning begins at the stem end and progressively affects the tissues, above mentioned, with varying severity toward the bud end (Fig. 1). The necrotic areas, so far as conspicuous browning is concerned, are not always continuous from the stem end on, but often discontinuous. In addition to the browned phloem strands of the vascular ring and of the outer and inner phloem regions, there are also frequently areas of irregular outline and of a water-soaked appearance in these regions, generally centering in the main vascular ring. The necrosis of the phloem seldom extends entirely to the bud end of the tuber.

The significance of the term "net necrosis" as descriptive of this trouble may be noted if the skin and outer cortex of the affected tuber are removed, when the branching and anastomosing strands of the outer phloem appear as a conspicuous brown network (Fig. 2).

In 1926 a favorable opportunity presented itself for studying the relation of net necrosis to leafroll, when an unusual outbreak of the disease occurred in the stock of a large producer in Vermont. In the grading of certain lots for spring shipment, 10 or 15 per cent of necrotic tubers were found. Several bushels of these tubers were secured, and selected necrotic tubers were planted in tuber units in an experimental plot. Other rows were planted

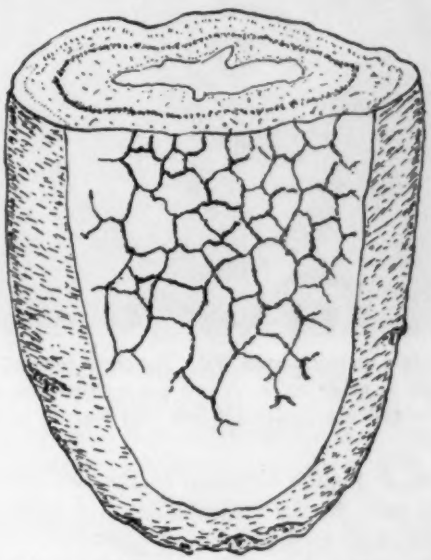

FIG. 2. Diagrammatic view of portion of necrotic tuber. The removal of epidermis and outer cortex reveals the brown, anastomosing strands of the necrotic phloem.

in tuber units with tubers as they came in the sacks, some necrotic and some free from necrosis. Still other rows contained units of healthy stock. An excellent opportunity was thus afforded to observe the progeny of these necrotic tubers in comparison with other stocks.

The necrotic tubers in tuber units produced practically 100 per cent leafroll hills (Fig. 3). In a total of two hundred hills there was not a single healthy unit and only a few hills where the leafroll symptoms were not clear. The rows planted with tubers as they came in the sacks, that is, without sorting out the necrotic ones, contained 25 per cent leafroll plants. This

FIG. 3. Experimental field at Burlington, Vt. The three rows at the right were planted with necrotic tubers in tuber units. There are many weak, and some missing, hills and practically 100 per cent leafroll units.

result, following similar results obtained in the two previous seasons, offers convincing evidence that, in the Green Mountain variety, there is an intimate relationship between the necrosis and leafroll.

Schultz and Folsom (8) mention "recency of infection" as one of the conditions associated with the appearance of net necrosis. This means that

necrosis is generally limited to tubers produced by plants the same season in which they have become infected with leafroll. According to Quanjer's definition (7), this is "primary" infection. Conspicuous leafroll symptoms may be present, or they may be entirely absent.

The observations of the present writer strongly indicate that net necrosis is a first-season symptom of leafroll following primary infection. In confirmation of this, many tubers from leafroll plants have been examined, both in the fall and at the close of the storage period, but well marked cases of necrosis have not been found. On the other hand, as stated above, necrotic tubers have almost invariably produced leafroll plants. This situation may explain why Atanasoff failed to find net necrosis upon the examination of several hundred leafroll tubers. In accordance with the above-mentioned observations, this was the result to be expected.

An examination of the illustrations of necrosis in Atanasoff's article would suggest that he might be picturing two distinct types of necrosis. The symptoms shown in his figure 2, in the variety Ashleaf, could hardly by any possibility be net necrosis of the phloem-necrosis type, as we understand it in America. In his illustration the necrotic areas do not follow the vascular ring, but are, in some of the slices, distinctly in the pith region, a condition which does not exist in phloem necrosis.

The tuber discolorations in the variety Roode Star, as shown in figure 3 of Atanasoff's paper, somewhat resemble net necrosis, but the subsequent history of the plants grown from such tubers would indicate that some other necrotic condition was present. The facts that the necrotic symptoms in the variety Roode Star were often confined to the stem end and that they were mistaken for Phytophthora lesions point strongly to the probability that the necrosis in these cases is distinct from the net necrosis as it occurs in the United States and as defined in the present paper.

Again, the correlation between the Holland necrosis of the tubers and aucuba mosaic of the vines makes it quite certain that the writer is dealing with a set of symptoms widely at variance with those associated with net necrosis in this country.

The occurrence of spindling sprout in connection with necrosis and leafroll has been observed repeatedly both by Schultz and Folsom (8, 9, 10) and by the writer (2). It is not stated that spindling sprout is a constant symptom of leafroll, but that it is of frequent occurrence in this connection, and, further, that it is quite consistently associated with net necrosis. It is often observed that some of the sprouts produced by a tuber in late storage are spindling, while others are normal. It will be noted in such cases that the spindling sprouts are toward the stem end, while the sprouts at the bud end may be normal. This does not mean the production of a healthy plant

from the terminal eyes, for the tuber acts as a unit in such cases in the production of leafroll plants. The plant or plants from the terminal buds will often be more vigorous than those from the eyes toward the basal end but will exhibit at least mild leafroll symptoms (Fig. 4). Further investigation will generally reveal that the part of the tuber producing the thin sprouts is affected with net necrosis. If necrosis of this type is at all severe in the tuber, the sprouts are almost certain to be weak and spindling.

FIG. 4. Tuber unit from necrotic tuber. The two hills at right are from basal eyes. All hills exhibited typical leafroll.

SUMMARY

The writer has endeavored to point out the facts that the term net necrosis has an accepted significance in the United States as applying to the browning and dying of the phloem tissues of the tuber, both outside and inside the cambium; that this necrosis has been found to be associated with leafroll, to the extent that necrotic tubers consistently produce leafroll plants and that net necrosis does not persist in leafroll stock but is believed to be a result of initial leafroll infection.

Both leafroll and net necrosis are generally associated with the production of spindling sprouts. Tubers seriously affected with necrosis will invariably produce spindling sprouts from a part or all of the eyes. If normal sprouts are produced by necrotic tubers, they will come from the eyes at the bud end, for the tissues in this region are seldom necrotic, but all plants produced will have symptoms of leafroll.

If the correlations between a potato tuber necrosis and aucuba mosaic, as described by Atanasoff, are substantiated, this will apparently be a second type of tuber necrosis of an infectious nature, the other being the phloem

necrosis associated with leafroll. The causal factors in connection with these diseased conditions are unknown.

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MADISON, WISCONSIN.

LITERATURE CITED

1. ATANASOFF, D. Net necrosis of potato. Phytopath. **16**: 929–940. 1926.
2. GILBERT, A. H. The correlation of foliage-degeneration diseases of the Irish potato with variations of the tuber and sprout. Jour. Agr. Res. **25**: 255–266. 1923. (Abstract in Phytopath. **12**: 40. 1922.)
3. KASAI, M. Observations and experiments on the leafroll disease of the Irish potato in Japan. Ohara Inst. Landw. Forsch. **2**: 47–77. 1921.
4. MCALPINE, D. Handbook of the diseases of the potato in Australia. Melbourne. 215 pp. 1911.
5. ORTON, W. A. Potato wilt, leafroll and related diseases. U. S. Dept. Agr. Bul. 64. 1914.
6. PAINE, S. G. Internal rust spot disease of the potato tuber. Rept. Internat. Conf. Phytopath. and Econ. Entom. Holland. p. 74. 1923.
7. QUANJER, H. M. Observations on "sprain" (Eisenfleckigkeit ? internal brown spot ?) and net necrosis of potatoes. Tijdschrift over Plantenziekten **32**: 97–128. 1926.
8. SCHULTZ, E. S., and DONALD FOLSOM. Leafroll, net necrosis and spindling sprout. Jour. Agr. Res. **21**: 47–80. 1921.
9. ———, ———. Transmission, variation and control of certain degeneration diseases of the Irish potato. Jour. Agr. Res. **25**: 43–117. 1923.
10. ———, ———. Infection and dissemination experiments with degeneration diseases of potato. Observations in 1923. Jour. Agr. Res. **30**: 493–528. 1925.
11. JONES, L. R., M. MILLER, and E. BAILEY. Frost necrosis of potato tubers. Wis. Agr. Exp. Sta. Research Bul. 46: 1–46. 1919.

A STUDY OF THE FUNGOUS FLORA OF THE NODAL TISSUES OF THE CORN PLANT[1]

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The nodal tissues of corn stalks often are found to be decomposed, even though the other parts of the plant may not be visibly affected. Such decomposition may be due either to microorganisms that are definitely parasitic or to other causes. Hoffer and Carr[2] and Hoffer and Trost[3] have quite conclusively demonstrated that, under certain field conditions, organic compounds of iron and aluminium accumulate in the nodal tissues of the corn plant. These accumulations interfere seriously with certain physiological activities of the plant, especially the translocation of foods and the conduction of food materials. As a result, the supply of foods to the root is interrupted and the whole plant is weakened. The leaves arising from affected nodes become fired, and eventually they are completely killed.

The firing of the lower leaves of the corn plant is a matter of common observation. The dead tissues of these leaves are invaded by various saprophytic fungi and bacteria. The organisms growing along and within these tissues arrive at and invade the tissues in the nodal region. The metal compounds account also for the killing of the roots, and thus another channel is provided for the entrance of saprophytes to the internal basal tissues. Such relations have been suggested by Hoffer and Trost.[3]

If the decomposition of nodal tissue is to be accounted for by the activities of definite parasites, such parasites should be isolated frequently from these tissues. On the other hand, if the organisms found in these tissues are secondary invaders, coming through the dead leaf tissues, a considerable variety of ordinary saprophytes should be found.

A study that would furnish reliable data toward the solution of such a problem would require the examination of a large number of nodes. This opportunity was provided during the summer of 1926. During this time

[1] Cooperative investigation of the Corn Disease Division of the Botany Department of the Agricultural Experiment Station and the Department of Biology, Purdue University.

[2] Hoffer, G. N., and R. H. Carr. Accumulation of aluminium and iron compounds in corn plants and its probable relation to root rots. I. Jour. Agr. Res. 23: 801–823. 1923.

[3] Hoffer, G. N., and J. F. Trost. Accumulation of iron and aluminium compounds in corn and its probable relation to root rots. II. Jour. Amer. Soc. Agron. 15: 323–331. 1923.

the Purdue Agricultural Experiment Station made a survey of the corn-producing areas of the East, South, and Middle West. This survey was for the primary purpose of determining the nutrient needs for corn in these areas by making chemical tests of the stalks. Portions of the corn stalks also were sent to the laboratory from each field surveyed. These stalks were cut off at the surface of the ground and consisted usually of five or six internodes. The age of the corn plants varied from the early roasting ear stage to full maturity. No plants were examined after freezing weather began.

It was my opportunity to culture the nodal tissues of the stalks sent to the laboratory. Stalks from approximately every fifth field surveyed were cultured. Cultures were made from stalks received from the following states: Alabama, Arkansas, Illinois, Indiana, Iowa, Kansas, Kentucky, Louisiana, Maine, Maryland, Michigan, Minnesota, Mississippi, Missouri, Nebraska, New York, Ohio, Oklahoma, Pennsylvania, South Dakota, Tennessee, Texas, Virginia, and Wisconsin. These stalks came from fields embracing many different soil types, fertility needs, and physical conditions, as well as a wide geographical and climatic range (Fig. 1).

The aims of the investigation set forth in this paper are: (a) to determine the flora resident in the nodal tissues of corn plants; (b) to ascertain whether the breakdown of nodal tissue is more frequently associated with the chemical and physical condition than with the presence of certain

FIG. 1. Map showing the localities from which corn stalks were examined.

specific organisms; (c) to determine if the chemical reserves of the stalk tissues have any appreciable effect on the kind of organisms present.

As soon as the stalks were received in the laboratory, they were split lengthwise. Half of the stalk was tested for the presence of nitrates, potassium, and iron. The methods used for making these tests are those described recently by Hoffer.[4] The color and state of decomposition of each of the nodal tissues tested were also recorded. A thick layer of the nodal tissues was removed from the other half-stalk with a sterile knife. This procedure exposed a sterile surface. Small pieces from each of the first four nodes were plated on potato dextrose agar under aseptic conditions in an inoculating chamber. For a time, pieces from opposite sides of the same node were plated to discover if there might be a difference in the growths resulting

TABLE 1.—*Organisms isolated from nodes of corn stalks collected from various parts of the United States in 1926. Arranged according to their relative percentage frequencies in isolations*

Organisms	No. of times isolated	Percentage of total no. of isolations
Type I Bacteria	659	28.30
Type III do	266	11.60
Mucor spp.	235	10.20
Fusarium spp.	232	10.05
Fusarium moniliforme	212	9.20
Type IV Bacteria	190	8.30
Penicillium spp.	141	6.10
Rhizopus nigricans	93	4.06
Aspergillus niger group	64	2.80
Yeasts	55	2.40
Type V Bacteria	49	2.10
Type VII do	47	2.05
Alternaria spp.	39	1.70
Type IV Bacteria	35	1.50
Diplodia zeae	28	1.20
Aspergillus glaucus group	19	0.83
Cephalosporium acremonium	17	0.70
Cephalothecium roseum	10	0.41
Type II Bacteria	7	0.35
Unidentified	7	0.35
Type VIII Bacteria	5	0.22
Helminthosporium gramineum	3	0.13
Aspergillus tamaraii group	2	0.09
Cylindrocephalum sp.	2	0.09

[4] Hoffer, G. N. Testing corn stalks chemically to aid in determining their plant food needs. Purdue University Agr. Exp. Sta. Bul. 298: 1–31. 1926.

from such bilateral plating. The two sides checked so frequently, however, that this line of procedure was abandoned as unnecessary. The total number of platings made was 2,097, and the total number of isolations was 2,288. Table 1 gives a tabulated list of organisms.

The average time that elapsed from the taking of stalks in the field until they were received in the laboratory was 48 hours. In order to determine the effect of holding stalks some time before plating, corn stalks were removed from a field near the Station and plated at once. Stalks from the same hills were wrapped in paper and held in the laboratory 72 hours before plating. In every case the two platings checked closely enough to indicate that delay in plating was a factor of very slight importance.

The types of bacterial organisms as given in table 1 are based on purely morphological characters. Each type is doubtlessly made up of a number of different species. These types may be characterized as follows:

Type I. Colony round, glistening white. Organisms 0.6×1.0 μ.

Type II. Colony dull, dry and wrinkled. Organisms 0.6×1.0 μ.

Type III. Colony light yellow. Organisms in chains. Spore formers with spores centrally located. 1.3×4 μ.

Type IV. Colony white, spreading "tree-like." Organisms 2.0×0.7 μ.

Type V. Colony egg-yellow, spreader. Organisms cocci. 0.6 μ in diameter.

Type VI. Colony glistening white, like the white of egg. Organisms spindle-shaped. 1.3×2.6 μ.

Type VII. Colony light-translucent in center and grayish white about border. Border crenate. Tendency to form gas in medium. Short heavy rod. 1.0×0.8 μ.

Type VIII. Wrinkled yellow colony. Organisms mixed, consisting of small rods 2.6×6 μ and long rods $14.3 \times .4$ μ.

Bacteria of these various sorts composed 50.7 per cent of the total organisms isolated.

There was no growth in 266 of the platings, or 12.7 per cent of the total number of platings were sterile. The distribution of sterile platings from the first, second, third, and fourth nodes is shown in table 2.

In table 2, Node I represents the lowermost nodes of the stalks plated. In practically every instance this node was the first visible node of the stalk above the surface of the ground. Nodes II, III, IV respectively are the nodes immediately above the lowermost node plated. Thus, as table 2 indicates, forty-five of the lowermost nodes plated were sterile. This number represents 2.14 per cent of all the platings made from the lowermost nodes. Of the total number of sterile platings made 16.92 per cent were from tissues of the lowermost nodes.

TABLE 2.—*The numbers and percentages of sterile platings from the first, second, third and fourth nodes of corn stalks collected from various parts of the United States in 1926*

Nodes	No. sterile platings	Percentage of total platings	Percentage of sterile plates
I	45	2.14	16.92
II	49	2.33	18.42
III	82	3.91	30.82
IV	90	4.29	33.83

It will be noted by reference to table 2 that the percentage of sterility increased progressively as the more superior nodal tissues of the stalk were tested, and that the number of sterile platings from the fourth node above the surface of the ground is exactly twice that from the lowermost node, or Node I.

These sterile nodes are significant especially if any reason may be assigned to their sterility. Hoffer and Carr[5] have shown that metallic accumulations in the nodes eventually cause the firing and death of the leaves arising from such nodes. It has also been shown that the content of iron and aluminium compounds in the nodal tissues is low when potassium salt reserves are present in the stalk tissues. A check was made of the sterile platings to note if there was any apparent relationship between the sterility of the nodal tissues and the iron and potassium accumulations. The results of this investigation are given in table 3.

TABLE 3.—*The relation of sterile platings to the iron accumulations of the nodes of corn stalks*

Iron accumulations[a]		No. sterile nodes	Percentage sterile nodes
Sub-normal	(C −)	51	19.17
Normal	(C)	145	54.51
	(C +)	40	15.03
Excessive	(D −)	13	4.88
	(D)	7	2.63
	(D +)	2	0.83
Very excessive	(E)	0	0.00
Unknown		8	3.00

[a] The letters used to denote the iron accumulations are the same as shown in Plate II, Bulletin 298 of the Purdue Agricultural Experiment Station. "C" denotes the quantity of iron in normal stalks; "D" denotes an excessive amount; and "E," the highest iron content found in disintegrated nodal tissues.

[5] Hoffer, G. N., and R. H. Carr. *Loc. cit.*

Examination of table 3 reveals the fact that nearly 90 per cent of all the sterile nodes carried an iron content that was practically normal, and also that there were very few sterile nodes to be found in stalks where the iron accumulations were much above those found in normal plants.

Stalks sent into the laboratory were tested to determine the presence of a potassium reserve. Potassium inhibits the accumulation of iron at the nodal tissues. A high potassium reserve in the stalk was invariably associated with a low iron accumulation. Of 67 stalks showing a potassium reserve of 1+ or more (slight excess), 50 were sterile. This relation suggests a correlation between potassium reserve, low iron accumulations, and sterility of the nodal tissues.

SUMMARY

A great variety of organisms, mostly saprophytic, constitutes the flora of the nodes of corn stalks.

A higher percentage of sterile platings was obtained from the upper nodal tissues than from the lower ones. This is associated with a greater firing of the lowermost leaves.

Sterile platings were more frequently made from nodes carrying a low iron content.

There was no close correlation between the kind of fungi in the nodal tissues and the soil type or degree of fertility of the soil, with the following exceptions: in plants containing a potassium reserve there was a greater tendency for the tissues to be healthy and sterile; and organisms almost always were found in tissues containing accumulations of iron.

No one organism, or several organisms, can be ascribed as primary agents in causing decomposition of nodal tissues.

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DUST TREATMENTS FOR THE CONTROL OF OAT SMUT IN IDAHO[1]

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Tests with dusts for the control of grain smuts were started at the Idaho Agricultural Experiment Station in 1920. These tests dealt, for the most part, with the control of bunt or stinking smut of wheat. The materials used were principally copper carbonate dust, together with a number of proprietary compounds. Of the materials used, copper carbonate has proved to be the most generally desirable disinfectant. This confirms work by other investigators.

Believing that some of these disinfectants might exhibit the same toxic property towards the seed-borne spores of oat smut, we conducted a group of tests in cooperation with the Crop Protection Institute. Some of the results were published in 1926.[2] It is sufficient to state that none of the dust treatments, nor the wet treatments with proprietary compounds, proved to be as effective in controlling oat smut as the formalin treatments.

No tests were conducted in 1925. Stimulated by the work of Thomas in Ohio, we continued the work again in 1926. These tests were conducted with both hulled and hulless oats. The test included twelve treatments and three untreated checks. Each treatment was replicated three times, each replication consisting of one rod row. Of the twelve treatments, nine were made with various dusts, with considerable emphasis placed upon mercuric chloride in combination with various types of fillers.

Unlike the results obtained by Thomas[3] or our own results of 1924, absolute control of smut of hulled oats was effected with copper carbonate, and there was only 0.33 per cent infection in the hulless variety. When mercuric chloride was used in combination with various fillers, in the proportion of one part filler to two parts mercuric chloride, complete control was secured in five out of six treatments. The sixth treatment allowed only 0.09 per cent infection. The indications are that mercuric chloride as a dust is quite effective. To what extent it can be diluted remains to be worked out.

These results are not in accord with the results of Thomas,[4] who failed to obtain control when kaolin was used as a filler, in combination with mercuric chloride alone, or with combinations of mercuric chloride and various

[1] Approved for publication by the Director of the Idaho Agricultural Experiment Station as Research Paper No. 46.

[2] Lambert, E. B., H. A. Rodenhiser, and H. H. Flor. The effectiveness of various fungicides in controlling the covered smuts of small grains. Phytopath. 16: 393–411. 1926.

[3] Thomas, R. C. Effective dust treatments for the control of smut of oats. Science 61: 47. 1925.

[4] Thomas, R. C. Control of smuts of wheat and oats with special reference to dust treatments. Ohio Agr. Exp. Sta. Bul. 390. 1925.

copper and nickel salts. We used ordinary Palouse silt loam as a filler. The soil was dried, pulverized with mortar and pestle, sifted through a 200-mesh sieve and mixed with mercuric chloride by hand. The only proportion used was two parts of mercuric chloride to one of filler. Thomas's investigations would seem to indicate that other proportions than the above are not effective.

Although the other dusts used controlled smut to a certain degree, none proved as efficient as either copper carbonate or mercuric chloride.

Table 1 gives the list of treatments and results obtained with both hulled

TABLE 1.—*Results of dust treatments for oat smut control with hulled and hulless oats in Idaho in 1926*

No.	Treatment	Results			
		Hulled oats		Hulless oats	
		Germination in per cent	Smut in per cent[a]	Germination in per cent	Smut in per cent[a]
1	Check. No treatment	99.0	7.52[b]	87.0	25.76[c]
2	Formalin (1–320) 5 min. Dip.	95.5	0.00	58.5	0.28
3	Formalin (1–1) spray, Cover 4 hrs.	100.0	1.02	7.5[d]	0.00
4	Formalin (1–10) spray, Cover 4 hrs.	95.0	0.00	4.5[d]	0.09
5	Copper, carbonate, 3 oz. per bu.	98.0	0.00	88.5	0.33
6	Copper carbonate, 1 part } 3 oz. Mercuric chloride, 2 parts } per bu.	99.0	0.00	85.0	0.00
7	Check. No treatment	99.5	12.49	83.5	9.60
8	Sulphur, 1 part } 3 oz. Mercuric chloride, 2 parts } per bu.	99.5	0.09	90.5	0.00
9	Palouse silt loam, 1 part } 3 oz. Mercuric chloride, 2 parts } per bu.	99.0	0.00	85.5	0.00
10	Dupont dust no. 49, 3 oz. per bu.	100.0	1.61	84.0	0.40
11	Dupont dust no. 52, 3 oz. per bu.	100.0	2.65	91.0	0.45
12	Semesan, 3 oz. per bu.	98.5	3.71	98.0	1.91
13	Bayer dust, 3 oz. per bu.	98.5	2.64	94.0	2.57
14	Colloidal copper, 3 oz. per bu.	98.0	4.93	97.5	2.32
15	Check. No treatment	98.5	14.45[c]	93.5	12.87

[a] Average of three replications.

[b] Average of one replication.

[c] Average of two replications.

[d] Much better germination was secured in field and a fair stand resulted.

and hulless oats. It will be noticed that germination of the hulless oats was seriously affected by the formalin treatments.

The tests reported upon here are but the results of one year's work (1926). Further tests are necessary before definite conclusions can be made.

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CHEMICAL HYDRATED LIME FOR THE PREPARATION OF BORDEAUX MIXTURE[1]

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The chief difficulty in preparing bordeaux mixture lies in the proper slaking of the lime. The suspension of the resulting calcium hydroxide and its activity or speed of reaction vary with the degree of dispersion or size of particles, as has been shown by different investigators. Unless the lime is thoroughly dispersed and free from large aggregates, the physical properties of the subsequent bordeaux will be seriously impaired and its efficiency as a fungicide reduced. The removal of coarse particles of lime and of insoluble matter from the milk of lime, by sieving or screening through double thickness of cheesecloth, is advisable in all cases but can not convert a poorly dispersed lime into a highly active product, to say nothing of the waste of material involved.

Everyone will concede that the hand-slaking of lime is hard to control, is laborious, and time-consuming, and that the substitution of commercial hydrated lime promises better and more uniform results provided a suitable product can be obtained at a reasonable cost. Agricultural lime is poorly adapted for the purpose; and ordinary plasterers' or finishing lime, containing 55 to 60 per cent active calcium hydroxide, carries too much inert material to be entirely satisfactory, although, if soaked in water over night before use, it yields a bordeaux of good suspension.

Chemical hydrated lime prepared from selected, high calcium limestone is now produced extensively in various sections of the country and is well suited for the preparation of bordeaux. In some instances the limestone employed contains substantially 99 per cent calcium carbonate and is burned in steel kilns fired by wood, coal, gas, or oil. The slaking may be done either by the batch system or the continuous flow process, and the resultant is generally air-separated (floated) to assure a light, bulky product free from grit. The material is white or yellowish, of fine particles, and of good flow unless damp, and, after proper soaking, of fair suspension and high activity. Data furnished by the manufacturers indicate that the better grades of such products contain from 93 to 98 per cent calcium hydroxide, 1.3 to 3.0 per cent calcium carbonate, and small amounts of ferric oxide, alumina, magnesia, sulphuric acid, silica and hygroscopic moisture. From 96.50 to 99.70 per cent will pass a 200-mesh screen. The bulk probably varies with the air-separation and subsequent treatment. Samples tested

[1] From the department of chemistry, Massachusetts Agricultural Experiment Station. Printed with the permission of the Director of the Station.

in the Massachusetts laboratory gave a volume of 2.4 to 3.3 cc. per gram, with an average of about 2.6 cc. The amount of active base in these samples, calculated as calcium hydroxide, averages about 89 per cent, with a range of 85 to 92 per cent. By active lime is meant that portion which can be readily determined by titration against standard acid to the initial disappearance of color, using thymolphthalein as indicator, thereby excluding coarse and other slowly reacting particles. The deterioration of chemical hydrated lime in paper bags, due to air-slaking (carbonating), is said to be less than that of quicklime in wooden barrels under similar conditions. Most producers ship the hydrate in paper bags holding 50 pounds; but cloth bags of 40 or 100 pounds, cloth and paper bags and wooden barrels of 180 pounds, and friction-top steel barrels are occasionally employed at a higher cost per unit. The cost in paper bags f. o. b. the plant varies from $10.00 to $15.00 per ton, and the freight to Amherst in l. c. l. lots is approximately the same amount, depending on the point of shipment.

Nine samples of chemical hydrated lime, representing deposits from Indiana, Missouri, Pennsylvania, Virginia, and West Virginia, were submitted by different manufacturers. Bordeaux 4–4–50 was prepared with each sample and the suspension determined as usual. The results are given in table 1.

TABLE 1.—*Suspension of bordeaux mixture (4–4–50) precipitated with chemical hydrated lime*

Chemical hydrated lime		Suspension in per cent		
Brand	Source	1 hr.	2 hrs.	3 hrs.
Lehigh	Mitchell, Ind.	97.2	93.8	90.4
Speed	Milltown, Ind.	97.9	94.5	91.7
Mississippi	Ste. Genevieve, Mo.	97.9	94.1	90.6
Peerless Special	do	97.4	93.3	90.2
Bell-Mine	Bellefonte, Pa.	96.8	92.7	88.9
Bell-Mine	do	97.7	94.1	90.7
Oranda	Capon Road, Va.	97.7	94.7	91.9
Berkeley	Berkeley, W. Va.	97.2	94.0	91.2
Super	Unknown	97.7	94.7	91.9
	Average.....	97.5	94.0	90.8

The quality of chemical hydrated lime depends on the purity of the deposit, the care exercised in mining and selection of the rock, and the chemical control of the manufacturing process. All the samples tested proved satisfactory for the preparation of bordeaux and can be recommended as desirable substitutes for quicklime.

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